DRAFT
HUMAN HEALTH RISK ASSESSMENT WORK PLAN

FOR THE
CAMP EDWARDS IMPACT AREA
GROUNDWATER QUALITY STUDY

MASSACHUSETTS MILITARY RESERVATION
CAPE COD, MASSACHUSETTS

Prepared for

NATIONAL GUARD BUREAU
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1. INTRODUCTION

This document presents a Work Plan for the human health risk assessment that will be completed as part of the Army National Guard's (ARNG) investigation of the Camp Edwards Training Range and Impact Area at the Massachusetts Military Reservation (MMR). This investigation is being undertaken in accordance with the Administrative Order on Consent SDWA I-97-1019, issued to the National Guard Bureau (NGB) by the Environmental Protection Agency (EPA) Region I on February 27, 1997. Currently, the groundwater underneath the northern portion of MMR is being considered for development as a potable water supply for the four upper Cape towns of Bourne, Falmouth, Mashpee, and Sandwich. Given this potential groundwater use, the purpose of the investigation is to study the effects of military operations on the groundwater beneath the Impact Area. An Action Plan prepared by the NGB (ETA, 1997) (see Section 1.1) provides procedures and guidance for gathering field data and information to determine whether there has been any impact to groundwater. The ARNG is working cooperatively with the EPA and Massachusetts Department of Environmental Protection (MADEP), the Long Range Water Supply Process Action Team (LRWSPAT), and the Cape Cod Commission, to complete this study.

The Action Plan specifies that both human health and ecological risk assessments be undertaken as part of the study. Ogden Environmental and Energy Services, Inc. (Ogden) will be the lead organization for performance of the human health risk assessment, and Los Alamos National Laboratory will be the lead organization for performance of the ecological risk assessment. This document addresses only the work plan for the human health risk assessment.

The stated objective of the study is to determine whether military operations have impacted groundwater beneath the Impact Area. Given this objective, the human health risk assessment will focus on the evaluation of potential health risk resulting from groundwater use as a potable water supply. In addition, the risk assessment will also evaluate potential human exposures to other environmental media within the Impact Area that may have been directly or indirectly impacted by military operations. As will be described in this Work Plan, potential exposures to surface soil within the Impact Area, and surface water and sediment in ponds within the Impact Area (that may be capable of supporting recreational activities) will also be evaluated in the human health risk assessment.

1.1 Study Background Information

In July 1996, the National Guard Bureau (NGB) and the Massachusetts Army National Guard (MAARNG) were directed by the Deputy Undersecretary of Defense for Environmental Security to study the effects of military operations on groundwater quality beneath the Camp Edwards Impact Area at the Massachusetts Military Reservation (MMR). In August 1996, the NGB assembled experts from the Army, the Air Force, MAARNG, the Army Corps of Engineers, the Army Environmental Policy Institute, Air Force Center for Environmental Excellence, Army Environmental Center, Army Center for Health Promotion and Preventive Medicine, and Georgia Institute of Technology, to create an Action Plan to complete the study.

An initial draft of the Action Plan was presented to U.S. EPA, the Massachusetts Department of Environmental Protection (MADEP) and the Cape Cod Commission (CCC) in December 1996. Following receipt of comments, a revised draft was issued in March 1997. The Administrative Order (SDWA I-97-1019) issued by EPA Region I directed the NGB to prepare the Action Plan for a comprehensive investigation of the groundwater beneath the Impact Area and provided for complete EPA oversight and participation. Subsequently, a draft final version of the Action Plan was prepared in consultation with EPA Region I and MADEP, and was submitted to EPA in May 1997 (ETA, 1997).

1.2 Site Description

Massachusetts Military Reservation (MMR) is a 21,000 acre facility located in the towns of Bourne, Falmouth, Mashpee, and Sandwich, Massachusetts. The facility is organized into four functional units:

- The Cantonment Area: The most actively used area, which consists of the base administrative, housing, maintenance, and operation facilities.
- The Training Range and Impact Area: Occupies most of the northern portion of the base and is used for military training, law enforcement training, and sport shooting.
- The Veterans National Cemetery: Located in the southwestern corner of MMR and contains a Veterans Administration Cemetery and support facilities.

- Cape Cod Air Force Station: Occupies the northeastern portion of the range area and houses the air defense warning system.

The Training Range and Impact Area, which is the focus of this investigation, occupies approximately 14,000 acres of MMR. This area is further broken down into the following:

- The Impact Zone. This was the target area for mortars and artillery and is therefore off limits to most personnel.
- The Buffer Zone. The Buffer Zone surrounds the Impact Zone and together they make up the Impact Area. Like the Impact Zone, access to the Buffer Zone is extremely limited.
- The Training Range Area. The Training Range surrounds the Impact Area. This area is used for arms training of MAARNG troops, and law enforcement officers. The small arms practice ranges are located in this area. Additionally, practice detonation of explosives takes place at two demolition ranges, one located in the northern portion and one at the southern edge of the Training Range Area. This area has also been used for fire protection training (ETA, 1997).

The relationship of these areas to each other are shown in Figure 1.

Munitions used at MMR include small arms munitions (e.g., 9 millimeter rounds), consisting of a lead core in a copper, iron, and antimony alloy jacket; artillery projectiles and mortar cartridges, similar to small arms but also include explosives; high explosives, such as TNT, RDX, mercury fulminate, and lead azide; and low explosives, such as black powder, smokeless powder, and other propellants (ETA, 1997). The explosives, as well as the metal projectiles from fired munitions, are the focus of the Training Range and Impact Area Groundwater Quality Study. In addition to evaluation of soil and groundwater conditions, several ponds and a swamp located within MMR will be included as a preliminary evaluation of the extent of constituent migration. Detailed information is provided in the Action Plan (ETA, 1997).

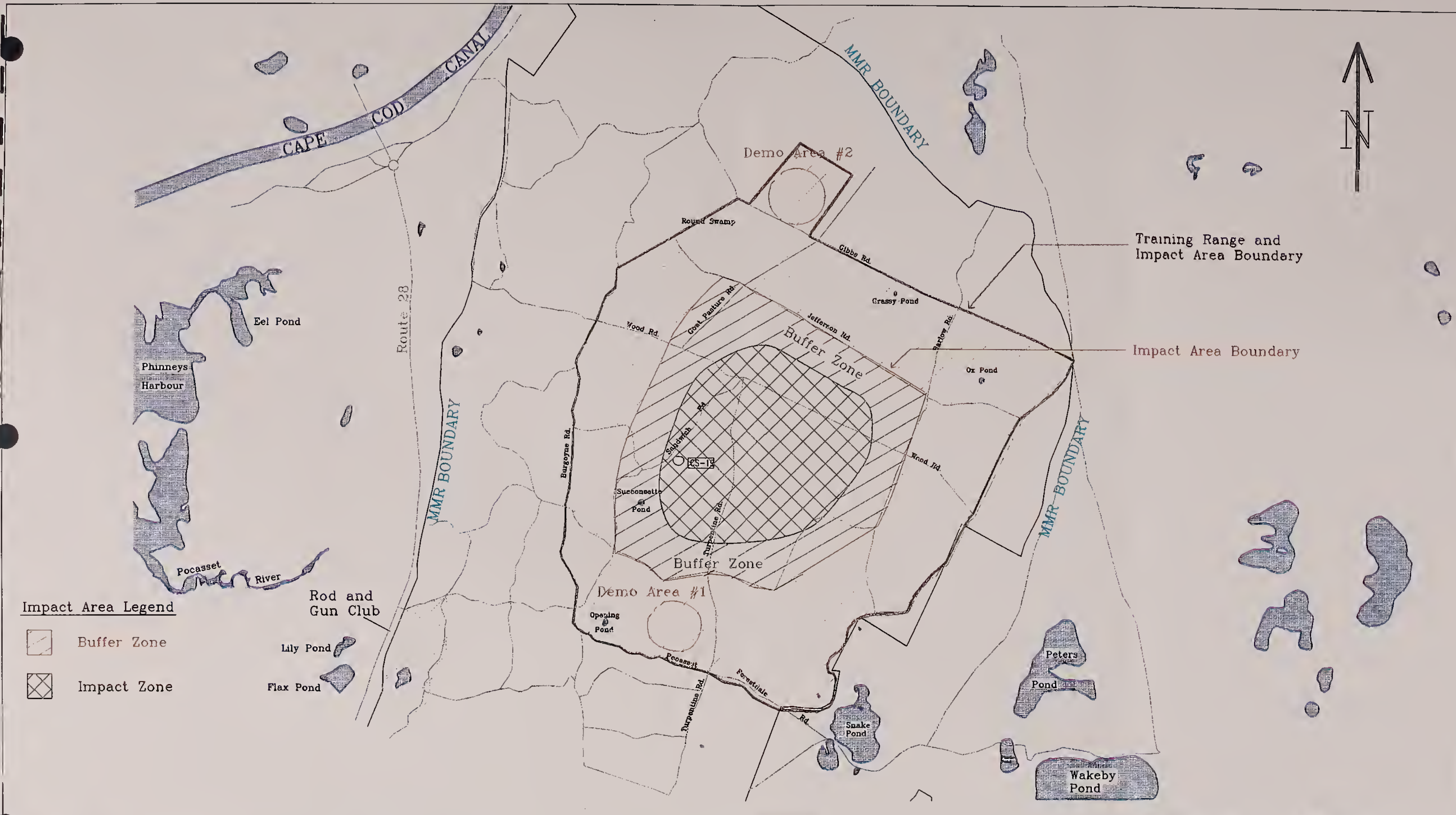
All firing training in the Training Ranges, with the exception of non-metallic small arms firing, has been suspended by EPA order. Depending on the outcome of the present study, activities in the study area may or may not be resumed.

1.3 Overview of the Risk Assessment Process

Risk Assessment of the Training Range and Impact Area will follow the procedures recommended in the *Final Risk Assessment Handbook, Volume I Risk Assessment Handbook, Massachusetts Military Reservation, Cape Cod, Massachusetts,* which was developed for the Installation Restoration Program at the MMR (IRP, 1994). The Risk Assessment Handbook (RAH) specifies a two-phased process.

The first phase of risk assessment is a Preliminary Risk Evaluation, which is discussed in Section 2 of this Work Plan. Briefly, the Preliminary Risk Evaluation serves two purposes: (1) determination of the need for additional study of the area and (2) determination if any immediate, interim action should be taken to prevent human exposures to high chemical concentrations during the course of study and selection of final remedies. In the event it is determined further study is required, one aspect of that study would be a site-specific quantitative risk assessment. Proposed methods for detailed quantitative risk assessment are discussed in Section 3 of this Work Plan. The RAH provides little guidance on how a site-specific quantitative risk assessment should be conducted. Therefore, the present Work Plan provides more definitive information on how this second-phase evaluation would be approached and employs both professional judgment and state and federal risk assessment guidance in addition to the RAH. The latter includes:

- *Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual.* (U.S. EPA, 1989);
- *Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors."* (EPA, 1991);
- *Dermal Exposure Assessment: Principles and Applications.*(U.S. EPA, 1992)
- *Background Documentation for the Development of the MCP Numerical Standards.* (MADEP, 1994); and,
- *Guidance for Disposal Site Risk Characterization - In Support of the Massachusetts Contingency Plan. Interim Final Policy.* (MADEP, 1995).



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MMR IMPACT AREA WORKPLAN

FIGURE 1
TRAINING RANGE AND IMPACT AREA

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2. PRELIMINARY RISK EVALUATION

As described in the RAH, a Preliminary Risk Evaluation (PRE) is conducted to determine the need for further study and to identify any immediate hazards requiring interim intervention prior to study completion. These determinations are made by comparing media-specific constituent concentrations observed during the study of the site to two separate risk-based concentrations developed specifically for the PRE. The comparison is made to individual sample observations, to provide a more conservative comparison than typical exposure point concentrations determined from multiple samples.

"Tier I" screening values are conservative risk-based values, which calculate a concentration that would protect at specified target risk levels for worst-case exposure assumptions. Below these concentrations it is concluded that estimated risk would be lower than that normally addressed with a remedial response, so that no further quantitative risk assessment is required. "Tier II" values are risk-based concentrations accounting for more realistic current exposures and with a higher target risk level. Tier II values, then, would be used for making a determination of the need for immediate intervention.

While the RAH lists certain Tier I and II values, the present Workplan proposes modifications for the following reasons:

- The RAH is currently being rewritten. It is Ogden's understanding that the revised RAH will do more to incorporate risk assessment procedures provided in guidance supporting both the Massachusetts Contingency Plan (MCP) and CERCLA actions (William Sweet, Brooks Air Force Base, May 15, 1997). Thus, Ogden is proposing PRE values that more completely acknowledge the risk assessment procedures associated with these programs. The most important aspect of Ogden's proposal is that MCP Reportable Concentrations be used for Tier I screening, where they are available.

- The existing RAH was developed for evaluation of discrete release areas and Areas of Concern identified during investigation of MMR, prior to the inclusion of the Impact Area as a location for environmental study. The exposure scenarios used to develop the Tier I and II values presented in the RAH are dependent upon the location of the particular study area, (i.e., with the Flightline Area or outside the Security Zone) (IRP,

1994) and thus, not necessarily relevant to the Training Range and Impact Area. As such, PRE values were calculated for area-specific exposures. Although site-specific exposure scenarios were developed, many of the exposure values (e.g., intake rates, exposure frequencies) used in these assumptions were obtained from the RAH (IRP, 1994).

Proposed Tier I and Tier II values are developed and reported in Appendix A to this report. Briefly, Ogden is proposing the use of MADEP Reportable Concentrations as Tier I screening values for groundwater and soils. These were selected because they assume a conservative exposure scenario (residential use of groundwater and soil), are based on target risk levels identical to those specified in the current RAH, and additionally make the preliminary risk assessment of these media similar to screening under the state hazardous waste site regulations known as the Massachusetts Contingency Plan (MCP). The Reportable Concentrations for organic compounds in soil account for leaching potential and thereby provide a somewhat broader PRE than those specified in the RAH. Tier I values for surface water and sediment were developed from exposure assumptions provided in the current RAH and elsewhere, and discussed in Section 5 of this Work Plan. Tier II values for each medium were developed using site-specific exposure scenarios, as discussed in Section 5 of this Work Plan, and applying higher risk targets, consistent with the requirements of the current RAH.

Tier I and Tier II values will be used as criteria for study decisions as specified in the *Response Matrix for the Camp Edwards Impact Area Groundwater Quality Study* (Ogden, 1997). Because the screening values are compared to individual observations, it is not necessary to complete all sampling and analysis prior to executing the PRE. Ogden will be distributing the analytical data to various parties very quickly, and will provide initial interpretation of the data by conducting the PRE for each data package prior to distribution.

Additionally, the Tier I values will be used to determine if a quantitative risk assessment, as described in subsequent Sections of this Work Plan, is required. In practice, this evaluation will be conducted by using the Tier I values as criteria for including a compound as a Constituent of Potential Concern in the detailed risk assessment.

3. SITE-SPECIFIC HUMAN HEALTH RISK ASSESSMENT

3.1 Hazard Identification

Chemical compounds that should be the subject of quantitative risk assessment (Constituents of Potential Concern; COPCs) will be selected from a candidate list comprised of any chemical compound detected in samples collected during the Impact Area Groundwater Quality Study. Media sampling and analysis planned for this study includes groundwater, soil (surface and subsurface), surface water, and sediment.

The objective of the Training Range and Impact Area Groundwater Quality Study is to determine whether activities have affected groundwater quality. Because the primary activity in these areas has been artillery and small arms training, the primary focus of the study includes propellants, explosives, and other munitions-related constituents. The subset of propellants, explosives, and their environmental degradation products that are of particular interest, as well as metals thought to be associated with site activities are listed in Table 1. In addition to analysis for explosives and metals, site samples will also be analyzed for volatile organics, semivolatile organics, pesticides/PCBs, and herbicides. Complete target compound/analyte lists are provided in the Action Plan (ETA, 1997). The following subsections discuss selection criteria for COPCs[1].

3.1.1 Selecting Compounds Based on Preliminary Risk Evaluation (PRE)

All detected constituents will be considered in the PRE. One outcome of the PRE will be a list of constituents of potential concern (COPCs), for all media, that will be included in a site-specific quantitative human health risk assessment. Any compound exceeding the risk target connoted by the PRE screening value will be considered for inclusion in the site-specific risk assessment.

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1 In the remainder of the Sections of this Work Plan, chemical data requirements are identified, with sources for the information. The actual acquisition of such data will not be undertaken until lists of COPCs are compiled, based on the selection criteria. However, both for illustrative purposes and to expedite EPA approval for risk assessment of chemicals most likely to be encountered, toxicity and chemical-specific exposure parameters are presented for the compounds of primary focus (Table 1).

Table 1
Constituents of Interest
Human Health Risk Assessment
Impact Area, Massachusetts Military Reservation

EXPLOSIVES	METALS
HMX	Antimony
RDX	Arsenic
1,3-Dinitrobenzene	Barium
1,3,5-Trinitrobenzene	Copper
Tetryl	Lead
Nitrobenzene	Mercury
2,4,6-Trinitrotoluene	Zinc
4-Amino-4,6-Dinitrotoluene	
2-Amino-2,6-Dinitrotoluene	
2,6-Dinitrotoluene	
2,4-Dinitrotoluene	
2-Nitrotoluene	
3-Nitrotoluene	
4-Nitrotoluene	
PETN	
Picric Acid	
Ammonium Picrate	

3.1.2 Site-Specific Background Conditions

Once all data for a particular study area is compiled, those constituents identified as candidate COPCs based on the PRE will be compared with site-specific background data. Constituents determined to be present at concentrations inconsistent with background concentrations (i.e., higher) will be selected as COPCs.

The Action Plan contains a proposed approach to establishing a site-specific background dataset for groundwater, soil, surface water, and sediment. Comparison of background to the site-specific dataset will be done using parametric inference on the means of background and site-data, where the datasets are found to be normally or log-normally distributed. A Shapiro-Wilks test will be used to evaluate the data distribution (Gilbert, 1987) and, if the data are found to fit a parametric distribution, a t-test will be used for inference on the means. If the data do not fit typical parametric distributions, a nonparametric, test, such as the Wilcoxon Rank Sum test (Gilbert, 1987) will be applied.

3.2 Dose-Response Assessment

The dose-response assessment identifies the relationship between the magnitude of COPCs to which a receptor is exposed (dose) and the likelihood of an adverse health effect (response). Both carcinogenic and noncarcinogenic health effects will be considered in the risk assessment.

3.2.1 Information Sources

Dose-response information (Reference Doses, RfD, for non-cancer effects and Cancer Slope Factors, CSF, for cancer evaluations) will be obtained from EPA's Integrated Risk Information System (IRIS) (EPA, 1997). If information is not available from IRIS, EPA's Health Effects Assessment Summary Tables (HEAST) (EPA, 1995) will be consulted as a secondary source. If dose-response information for a COPC is not available from any of these sources, then other sources, including MADEP's Office of Research and Standards (ORS) and the Agency for Toxic Substances Disease Registry (ATSDR) will be reviewed for suitable information All toxicity values will be presented and documented in the risk assessment report. Where no source of data is identified, Ogden will suggest toxicity factors

for certain compounds, to the extent possible, for the evaluation and approval of EPA Region I risk assessment specialists. The suggestions will be based on the best professional judgment of a toxicologist, who will evaluate compounds with respect to compounds of similar chemical structure for which toxicity data are available.

Table 2 provides toxicity factors for the compounds of primary focus, in order to illustrate the information acquisition procedure. It can be seen that while significant data could be obtained from recognized sources, certain professional judgments may have to be made to complete risk assessment of the environmental breakdown products of explosive and propellants, should they be detected. In addition to recognized sources, these include:

- An EPA memorandum (Harry Craig, EPA Oregon Operations to Jane Dolan, Region I by facsimile on 4/23/97) suggests the use of an RfD for Tetryl of 0.05 mg/kg-day and CSFs for 2,4- and 2,6-dinitrotoluene of 0.68 kg-day/mg.[2] Also suggested in this memorandum is the use of an RfD of 0.003 mg/kg-d for picric acid. Ogden proposes to use this RfD for picric acid and for ammonium picrate, as there are no RfDs available from IRIS or HEAST and because they are structurally similar.
- Additionally, Ogden could find no information on the nitro reduction products of 2,4,6, trinitrotoluene (TNT), which include 2-amino-4,6 dinitrotoluene, 4-amino-2,6-dinitrotoluene, 2,6-diamino-4 nitrotoluene, and 2,4-diamino 6-nitrotoluene. We suggest, based on the structural similarity of the reduction products to the parent TNT, that the RfD and CSF for TNT be used for all of the compounds. To the extent that reduction of the nitro- groups makes the molecules less reactive, we suspect the assumption of equal carcinogenic potency is conservative.
- Lastly, no information was found for PETN (pentaerythritol tetranitrate) and no suitable surrogates were identified at this time. Ogden will confer with EPA Region I to determine an appropriate toxicity factor for this compound, should it become necessary.

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[2] Ogden assumes the CSFs derive from an IRIS citation for "mixtures" of the 2,4 and 2,6 isomers of dinitrotoluene, because CSFs for the individual isomers are not listed in IRIS. The use of this CSF for both isomers is conservative, because the experiment underlying the dose-response value utilized a 98% mixture of 2,4-dinitrotoluene with only 2% 2,6-dinitrotoluene (Indeed the RfD listed in IRIS for 2,4-dinitrotoluene alone is based on an experiment using the same ratio of 2,4- and 2,6 dinitrotoluene). Thus, it is questionable if 2,6-dinitrotoluene has ever been adequately tested for carcinogenicity.

Table 2
Toxicity Information for Constituents of Interest
Human Health Risk Assessment
Target Range and Impact Area, Massachusetts Military Reservation

Compound	Oral RfD Chronic mg/kg/day	Oral RfD Source	Inhalation RfD Chronic mg/kg/day	Inhalation RfD Source	Oral Slope Factor $(mg/kg/day)^{-1}$	Source	Inhalation Slope Factor $(mg/kg/day)^{-1}$	Source
EXPLOSIVES								
HMX	0.05	IRIS, 6/97	0.05	Oral RfD	NA		NA	Oral Slope Factor
RDX	0.003	IRIS, 6/97	0.003	Oral RfD	0.11	IRIS, 6/97	0.11	Oral Slope Factor
1,3-Dinitrobenzene	0.0001	IRIS, 6/97	0.0001	Oral RfD	NA		NA	Oral Slope Factor
1,3,5-Trinitrobenzene	0.00005	IRIS, 6/97	0.00005	Oral RfD	NA		NA	Oral Slope Factor
Tetryl	0.05	EPA Memorandum 4/97	0.05	Oral RfD	NA		NA	Oral Slope Factor
Nitrobenzene	0.0005	IRIS, 6/97	0.0006	HEAST, 5/95[2]	NA		NA	Oral Slope Factor
2,4,6-Trinitrotoluene (TNT)	0.0005	IRIS, 6/97	0.0005	Oral RfD	0.03	IRIS, 6/97	0.03	Oral Slope Factor
2-Amino-4,6-Dinitrotoluene	0.0005	use TNT as surrogate	0.0005	Oral RfD	0.03	use TNT as surrogate	0.03	Oral Slope Factor
4-Amino-2,6-Dinitrotoluene	0.0005	use TNT as surrogate	0.0005	Oral RfD	0.03	use TNT as surrogate	0.03	Oral Slope Factor
2,6-Dinitrotoluene	0.001	HEAST, 5/95	0.001	Oral RfD	0.68	IRIS, 6/97	0.68	Oral Slope Factor
2,4-Dinitrotoluene	0.002	IRIS, 6/97	0.002	Oral RfD	0.68	EPA Memorandum 11/91	0.68	Oral Slope Factor
2-Nitrotoluene	0.01	HEAST, 5/95	0.01	Oral RfD	NA		NA	Oral Slope Factor
3-Nitrotoluene	0.01	HEAST, 5/95	0.01	Oral RfD	NA		NA	Oral Slope Factor
4-Nitrotoluene	0.01	HEAST, 5/95	0.01	Oral RfD	NA		NA	Oral Slope Factor
2,6-Diamino-4-nitrotoluene	0.01	4-Nitrotoluene surrogate	0.01	Oral RfD	NA		NA	Oral Slope Factor
2,4-Diamino-6-nitrotoluene	0.01	4-Nitrotoluene surrogate	0.01	Oral RfD	NA		NA	Oral Slope Factor
PETN								
Picric Acid	0.003	EPA Memorandum 4/97	0.003	Oral RfD	NA		NA	Oral Slope Factor
Ammonium Picrate	0.003	Picric Acid surrogate	0.003	Oral RfD	NA		NA	Oral Slope Factor
INORGANICS								
Antimony	0.0004	IRIS, 6/97	0.0004	Oral RfD	NA		NA	Oral Slope Factor
Arsenic	0.0003	IRIS, 6/97	0.0003	Oral RfD	1.5	IRIS, 6/97	15.1	IRIS, 6/97
Barium	0.07	IRIS, 6/97	0.0001	HEAST, 5/95[2]	NA		NA	Oral Slope Factor
Copper	0.037	HEAST, 5/95[1]	0.037	Oral RfD	NA		NA	Oral Slope Factor
Mercury	0.0003	IRIS, 6/97	8.57E-05	IRIS, 6/97[2]	NA		NA	Oral Slope Factor
Zinc	0.3	IRIS, 6/97	0.3	Oral RfD	NA		NA	Oral Slope Factor

Notes:

1. The Oral RfD for copper was calculated from the MCL of 1.5 mg/L, assuming ingestion of 2 L/day by a 70 kg adult (1.5 mg/L *2 L/d / 70 kg = 0.037 mg/kg-d).
2. The Inhalation RfDs for nitrobenzene, barium and mercury were calculated from the RfCs obtained from HEAST or IRIS, assuming an inhalation rate of 20 m^3/day by a 70 kg adult (RfC mg/m^3 * 20 m^3/day / 70 kg = RfD mg/kg-day).

IRIS: U.S. EPA's Integrated Risk Information System. On-Line Database. Information verified 6/97.
HEAST: U.S. EPA's Health Effects Assessment Summary Tables, FY-1995 Annual. EPA/540/R-95/036.
EPA Memorandum 4/97. Memorandum from Harry Craig, EPA Oregon Operations to Jane Dolan, Region I re: RBC Tables and Cleanup Level Summary. by facsimile.

3.2.2 The Special Case of Lead

Unlike most compounds whose health risk is calculated as a function of administered dose, lead toxicity has typically been reported as a function of blood lead concentration. U.S. EPA has generally expressed a goal of protecting at least 95% of small children exposed to lead below a blood lead level of 10 micrograms per deciliter (10 ug/dL), but has not specified a protective goal in adults. However, U.S. EPA has recently suggested that evaluation of lead exposures in adults should be evaluated based on the concept of protecting the potential fetus in women of child-bearing age (U.S. EPA, 1996). In this guidance, U.S. EPA used a blood lead goal in fetuses of 10 ug/dL, a value that will be used for the purposes of this Work Plan.

3.2.3 Relative Absorption Factors

Relative absorption factors (RAFs) will be used in this risk assessment, consistent with EPA and MADEP risk assessment approaches. RAFs are necessary to adjust estimated exposure doses (discussed in Section 5) to be compatible with the bioavailability of doses given in the experiments that form the basis of the toxicity factors. This is required because the efficiency of COPC absorption via a particular route and matrix being evaluated (e.g., ingestion of soil) varies from the absorption efficiency for the exposure route and matrix used in the experimental study that is the basis of the toxicity value. RAFs will be presented and documented in the risk assessment report. EPA Region I has developed default RAFs for particular chemical classes (U.S. EPA Region I, 1989), which will be used where chemical specific information is unavailable.

Table 3 lists RAFs for different exposure media and routes of exposure for the constituents of primary focus in the Training Range and Impact Area Groundwater Quality Study, as an illustration of procedure. No information was available on the absorption efficiency of propellants, explosives, and degradation products and, as such, Region I default RAFs are indicated for all routes and media for which they were available. For the most part, defaults were also used for metals, with the exception of RAFs based on information obtained by Ogden for arsenic, mercury and zinc. Appendix B to this report describes the studies supporting the proposed RAFs listed in Table 3. No default values or experimental data are available for RAFs for the dermal-water exposure route. In this case, Ogden assumed dermal uptake and oral bioavailability were equal.

Table 3
Relative Absorption Factors for Constituents of Interest
Human Health Risk Assessment
Impact Area, Massachusetts Military Reservation

	ORAL-SOIL/SEDIMENT		DERMAL-SOIL/SEDIMENT		ORAL-WATER		DERMAL-WATER	
Compound	RAF	Source	RAF	Source	RAF	Source	RAF	Source
EXPLOSIVES								
HMX	1	EPA Region I (1)	0.5	EPA Region I (1)	1	EPA Region I (1)	1	No data
RDX	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
1,3-Dinitrobenzene	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
1,3,5-Trinitrobenzene	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
Tetryl	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
Nitrobenzene	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
2,4,6-Trinitrotoluene	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
2-Amino-4,6-Dinitrotoluene	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
4-Amino-2,6-Dinitrotoluene	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
2,6-Dinitrotoluene	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
2,4-Dinitrotoluene	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
2-Nitrotoluene	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
3-Nitrotoluene	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
4-Nitrotoluene	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
2,6-Diamino-4-nitrotoluene	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
2,4-Diamino-6-nitrotoluene	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
PETN	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
Picric Acid	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
Ammonium Picrate	1	EPA Region I	0.5	EPA Region I	1	EPA Region I	1	No data
INORGANICS								
Antimony	1	EPA Region I	0	EPA Region I	1	EPA Region I	1	No data
Arsenic	0.41	Ogden (2)	0	EPA Region I	1	EPA Region I	1	No data
Barium	1	EPA Region I	0	EPA Region I	1	EPA Region I	1	No data
Copper	1	EPA Region I	0	EPA Region I	1	EPA Region I	1	No data
Mercury	2	Ogden (2)	0	EPA Region I	2	Ogden (2)	13.7	Ogden (2)
Zinc	1	EPA Region I	0	EPA Region I	1.6	Ogden (2)	3.03	Ogden (2)

Notes:
1. U.S. EPA, Region I. 1989. Supplemental Risk Assessment Guidance for the Superfund Program. Draft Final. EPA/901/5-89-001. June. Suggests and RAF of 1 for oral exposure routes and, for dermal absorption from soil a value of 50% for semivolatiles (applied here to the explosive, propellants and degradation products) and "negligible" (assumed to be zero) for metals.
2. Ogden values derived by staff toxicologists. Background documentation is provided in Appendix B.
3. Dermal-water RAFs of 1 were assigned to all chemicals, except antimony, mercury, and zinc, based on a lack of data.

3.3 Exposure Assessment

This section presents a conceptual exposure model developed for the Impact Area Groundwater Quality Study. This conceptual model consists of a description of activities and uses associated with the Impact Area and the exposure potential related to these activities. The conceptual model was developed based on available information regarding current and foreseeable site use and conditions. This section also presents the methodology and equations that will be used to calculate exposure point concentrations and potential exposure dose.

3.3.1 Potential Human Receptors

A "receptor" is a hypothetical individual who may be exposed to compounds in the environment, currently or in the future. Receptors are often identified by the behaviors that determine how or with what intensity they may be exposed. The following section describes receptors to be evaluated in the risk assessment of the Training Range and Impact Area. A summary of potential receptors is provided in Table 4.

3.3.1.1 Residential Groundwater Users

Groundwater use as a potable water supply will be evaluated and offsite residents potentially using groundwater are identified as a current and future receptor, for the following reasons. There are no permanent buildings or other structures located within the Impact Area and therefore, there is no onsite groundwater use within this area. However, groundwater in the northern portion of MMR is being considered for development as a potable water supply for the four towns of Bourne, Falmouth, Mashpee, and Sandwich. In fact, the potential for groundwater withdrawal for domestic use from locations closer to the Training Ranges and Impact Area for offsite domestic use has been adequately demonstrated by the water supply studies being conducted in the vicinity. It is possible that these towns may initiate this groundwater use in the near future (i.e., within one or two years).

Table 4
Potential Exposure Pathways
Human Health Risk Assessment
Impact Area, Massachusetts Military Reservation

Receptor	Age	Exposure Point	Medium	Exposure Route
CURRENT AND FUTURE SITE USE				
Off-Site Residents	Child/Adult (age 1 - 30)	Off-Site Residence	Groundwater in Impact Area (water supply)	Ingestion
ANG Personnel	Adult (age >18)	Individual Shooting Ranges	Soil (0 - 1 ft.)	Incidental Ingestion Dermal Contact Inhalation of Soil-Derived-Dust
Law Enforcement Personnel	Adult (age >18)	Individual Shooting Ranges	Soil (0 - 1 ft.)	Incidental Ingestion Dermal Contact Inhalation of Soil-Derived-Dust
Tresspasser/ Recreational	Older Child/Adult	Entire Training Range and Impact Area	Soil (0 - 1 ft.)	Incidental Ingestion Dermal Contact Inhalation of Soil-Derived-Dust
Tresspasser/ Recreational	Older Child/Adult	Ponds	Surface Water	Incidental Ingestion Dermal Contact
			Sediment	Incidental Ingestion Dermal Contact

3.3.1.2 Trespassers/Recreational Users

Trespassers have been observed in the Impact Area. Access to the Impact Area is extremely restrictive and no training activities take place here. Authorized personnel (e.g., range control officers) are rarely on foot in the area, nor do they have frequent access to area media. Personnel involved in the Impact Area Groundwater Quality study and unexploded ordnance clearance specialists operate under a health and safety plan requiring personal protective equipment, which largely eliminates exposures. Thus, individuals trespassing on foot, may currently be the receptor who would be most exposed to material in the Impact Area. While no groundwater use would be anticipated for these individuals, exposures might occur as the result of contact with materials in soil (ingestion dermal absorption, inhalation of entrained dust or vapor releases). Additionally, it is possible that trespassers may be in the area of the ponds to be investigated as a supplement to the Impact Area study. Exposure via swimming or wading will thus also be addressed for the trespasser. Only some of the ponds within the MMR may be large enough to support such activities; intermittent ponds and vernal pools will not be considered for this exposure.

Because the ARNG has no plans to change the use of the study area, the most reasonable foreseeable future use will be the same as current use, which would cause the Trespasser to also be a receptor in the future. However, it might also be possible to use the area as conservation or recreational space. Indeed a deer hunt for purposes of herd control is periodically held on the MMR. It seems improbable that residential development of the area would occur, given the potential for the area to be developed as a groundwater supply. Based on this a reasonable maximal exposure could also include recreational uses of the property. By using reasonably conservative exposure parameters for the trespasser, such a recreational receptor could also be accounted for. This approach is proposed here.

3.3.1.3 ARNG and Law Enforcement Personnel

Target shooting and most firing training activities have been discontinued, thus, there is limited activity currently taking place in the Training Ranges. However, ARNG was training in this area until recently and law enforcement personnel (e.g., Massachusetts State Police) continue small arms training in the Training Range. Thus, this receptor is appropriate for

evaluation of current exposure, particularly in the Training Range areas. ARNG anticipates continuing training in the future, so that this receptor is also proposed as a potential future receptor. Exposure in such receptors would be limited to direct contact with soil (ingestion, dermal absorption, inhalation of entrained dusts or vapors).

3.3.2 Potential Exposure Pathways

Exposure pathways are the mechanisms by which receptors may be potentially exposed to constituents of concern. According to EPA (1989), an exposure pathway consists of the following elements:

- a source and mechanism by which a constituent may be released into the environment;
- an environmental transport medium or mechanism of transport from one medium to another;
- a potential point of contact with the affected medium (exposure point); and,
- a potential receptor and route of exposure at the exposure point.

The potential exposure pathways that will be evaluated for each identified receptor are listed in Table 4. Note that several receptors and exposure pathways are assumed to be identical for present and future use. This is either because a) the receptor is anticipated to be identical (e.g., ARNG, law enforcement personnel) or, b) the behaviors of current and future receptors are anticipated to be roughly similar (e.g., current trespasser and future recreational receptor), resulting in similar exposures.

3.3.2.1 Groundwater

Potential exposure to groundwater as a potable water supply will be quantitatively evaluated via ingestion only. Dermal contact with constituents in groundwater (i.e., while showering) will not be evaluated because of the short duration and, hence, low magnitude, of this potential exposure. Evaluation of inhalation exposure to constituents in groundwater while showering is not currently contemplated given the short duration of this potential exposure and the low or non-volatility of the constituents of primary focus (propellants, explosives, and metals). However, if volatile COPCs are identified in the PRE, the exposure potential via this pathway will be considered.

3.3.2.2 Soil, Surface Water, and Sediment

For all receptors potentially exposed to site soils, exposure via incidental ingestion, dermal contact, and inhalation of soil-derived will be quantitatively evaluated. Surface water and sediment exposures via incidental ingestion and dermal contact will also be quantitatively evaluated.

3.3.3 Exposure Parameters

Table 5 lists the exposure parameters anticipated for the identified receptors at this point in time, although Ogden will continue to study activities in the Impact Area and revise exposure parameters as needed.

EPA-recommended values will be used for exposure parameters such as groundwater consumption rate, soil ingestion rate, and surface water contact rate. For site-specific exposure parameters such as exposure frequency and duration, site-specific values (based on information received from the ARNG) will be used.

It is notable that certain receptors are anticipated to be exposed in similar areas. As such, the most exposed of the various receptors in an area is of the greatest interest, because if one makes risk management decisions based on the most exposed receptor, they will also be protective of other receptors. Therefore, if Ogden chooses only one receptor for an area, it will be the hypothetically most exposed receptor identified in that exposure area. Similarly, note that certain PRE screening values (see Section 2) were developed from the exposure scenarios described here. As noted in Appendix A to this Work Plan, the screening values were derived based on the most exposed receptor for each area and medium.

3.3.4 Exposure Point Concentrations

Consistent with EPA guidance, exposure point concentrations for all media, except groundwater will be the 95% upper confidence limit (UCL) on the mean concentration within an exposure area. For groundwater, arithmetic mean and maximum concentrations will be

Table 5
Exposure Parameters
Human Health Risk Assessment
Impact Area, Massachusetts Military Reservation

RECEPTOR	PATHWAY/EXPOSURE FACTOR		COMMENTS
Offsite Resident: Child	**Ingestion of Groundwater**		
	Ingestion Rate (L/day)	1	U.S. EPA, 1991.
	Exposure Frequency (days/yr)	350	U.S. EPA default residential exposure frequency (U.S. EPA, 1991).
	Exposure Duration (yr)	6	assume 6 yrs. of residential exposure is as a child (U.S. EPA, 1991).
	Body Weight (kg)	16	avg. for males and females age 1 - 6 (U.S. EPA, 1990).
	Averaging Time - Noncancer (days)	2,190	exposure duration * 365 days/year.
	Averaging Time - Lifetime (days)	25,550	70-yr lifetime exposure duration * 365 days/year.
Offsite Resident: Adult	**Ingestion of Groundwater**		
	Ingestion Rate (L/day)	1	U.S. EPA, 1991.
	Exposure Frequency (days/yr)	350	U.S. EPA default residential exposure frequency (U.S. EPA, 1991).
	Exposure Duration (yr)	24	assume 24 yrs. of residential exposure is as an adult (U.S. EPA, 1991).
	Body Weight (kg)	70	average adult body weight (U.S. EPA, 1990).
	Averaging Time - Noncancer (days)	8,760	exposure duration * 365 days/year.
	Averaging Time - Lifetime (days)	25,550	70-yr lifetime exposure duration * 365 days/year.
ANG Personnel	**Incidental Ingestion of & Dermal Contact with Soil**		
	Ingestion Rate (mg/day)	50	U.S. EPA, 1991.
	Fraction from Site (unitless)	1	assume, on days exposed, all soil exposure occurs at the site.
	Surface A.ea Exposed (cm^2/day)	3,700	surface area of adult male hands, forearms, and head (U.S. EPA, 1990).
	Soil-to-Skin Adherence Factor (mg/cm^2)	1	upper bound value (U.S. EPA, 1992).
	Exposure Frequency (days/yr)	38	one weekend per month, plus two full weeks.
	Exposure Duration (yr)	25	U.S. EPA default occupational exposure duration (U.S. EPA, 1991).
	Body Weight (kg)	70	average adult body weight (EPA, 1990).
	Averaging Time - Noncancer (days)	9125	exposure period * 365 days/year.
	Averaging Time - Lifetime (days)	25,550	70-yr lifetime exposure period * 365 days/year.
ANG Personnel	**Inhalation of Fugitive Dust**		
	Inhalation Rate (m^3/hr)	2.5	20 m^3 per 8 hour workday (U.S. EPA, 1991).
	Particulate Emission Factor (m^3/kg)	1.32E+09	EPA, 1996.
	Exposure Time (hr/day)	8	assume dust is generated throughout the work day.
	Exposure Frequency (days/yr)	38	one weekend per month, plus two full weeks.
	Exposure Duration (yr)	25	U.S. EPA default occupational exposure duration (U.S. EPA, 1991).
	Body Weight (kg)	70	average adult body weight (EPA, 1990).
	Averaging Time - Noncancer (days)	9125	exposure period * 365 days/year.
	Averaging Time - Lifetime (days)	25,550	70-yr lifetime exposure period * 365 days/year.
Law Enforcement	**Incidental Ingestion of & Dermal Contact with Soil**		
Personnel	Ingestion Rate (mg/day)	50	U.S. EPA, 1990.
	Fraction from Site (unitless)	1	assume, on days exposed, all soil exposure occurs at the site.
	Surface Area Exposed (cm^2/day)	3,700	surface area of adult male hands, forearms, head (U.S. EPA, 1990).
	Soil-to-Skin Adherence Factor (mg/cm^2)	1	upper bound value (U.S. EPA, 1992).
	Exposure Frequency (days/yr)	12	once per month.
	Exposure Duration (yr)	25	U.S. EPA default occupational exposure duration (U.S. EPA, 1991).
	Body Weight (kg)	70	average adult body weight (EPA, 1990).
	Averaging Time - Noncancer (days)	9125	exposure period * 365 days/year.
	Averaging Time - Lifetime (days)	25,550	70-yr lifetime exposure period * 365 days/year.
Law Enforcement	**Inhalation of Fugitive Dust**		
Personnel	Inhalation Rate (m^3/hr)	2.5	20 m^3 per 8 hour workday (U.S. EPA, 1991).
	Particulate Emission Factor (m^3/kg)	1.32E+09	EPA, 1996.
	Exposure Time (hr/day)	8	assume dust is generated throughout the work day.
	Exposure Frequency (days/yr)	12	once per month.
	Exposure Duration (yr)	25	U.S. EPA default occupational exposure duration (U.S. EPA, 1991).
	Body Weight (kg)	70	average adult body weight (EPA, 1990).
	Averaging Time - Noncancer (days)	9125	exposure period * 365 days/year.
	Averaging Time - Lifetime (days)	25,550	70-yr lifetime exposure period * 365 days/year.

Table 5
Exposure Parameters
Human Health Risk Assessment
Impact Area, Massachusetts Military Reservation

RECEPTOR	PATHWAY/EXPOSURE FACTOR		COMMENTS
Trespasser/Recreational Receptor	**Incidental Ingestion of & Dermal Contact with Soil**		
	Ingestion Rate (mg/day)	50	U.S. EPA, 1991.
	Fraction f:o n Site (unitless)	1	assume, on days exposed, all soil exposure occurs at the site.
	Surface Area Exposed (cm^2/day)	3,700	surface area of adult male hands, forearms, head (U.S. EPA, 1990).
	Soil-to-Skin Adherence Factor (mg/cm^2)	1	upper bound value (U.S. EPA, 1992).
	Exposure Frequency (days/yr)	24	assume trespassing/recreation occurs 2 days/month, year round.
	Exposure Duration (yr)	30	U.S. EPA default residential exposure duration (U.S. EPA, 1991).
	Body Weight (kg)	70	average adult body weight (EPA, 1990).
	Averaging Time - Noncancer (days)	10,950	exposure period * 365 days/year.
	Averaging Time - Lifetime (days)	25,550	70-yr lifetime exposure period * 365 days/year.
Trespasser/Recreational Receptor	**Inhalation of Fugitive Dust**		
	Inhalation Rate (m^3/hr)	2.0	average for adults at moderate activity level (U.S. EPA. 1990).
	Particulate Emission Factor (m^3/kg)	1.32E+09	EPA, 1996.
	Exposure Time (hr/day)	4	assume 1/2 day spent in the Impact Area when trespassing.
	Exposure Frequency (days/yr)	24	assume trespassing/recreation occurs 2 days/month, year round.
	Exposure Duration (yr)	30	U.S. EPA default residential exposure duration (U.S. EPA, 1991).
	Body Weight (kg)	70	average adult body weight (EPA, 1990).
	Averaging Time - Noncancer (days)	10,950	exposure period * 365 days/year.
	Averaging Time - Lifetime (days)	25,550	70-yr lifetime exposure period * 365 days/year.
Trespasser/Recreational Receptor	**Incidental Ingestion of & Dermal Contact with Surface Water**		
	Ingestion Rate (ml/hr)	50	U.S. EPA, 1990; IRP, 1994.
	Surface Area Exposed (cm^2/day)	19,400	average adult total body surface area (U.S. EPA, 1990; IRP, 1994).
	Exposure Time (hr/day)	2.6	IRP, 1994.
	Exposure Frequency (days/yr)	7	IRP, 1994.
	Exposure Duration (yr)	30	Default residential exposure duration (U.S. EPA, 1991; IRP, 1994).
	Body We'ght (kg)	70	average adult body weight (EPA, 1990; IRP, 1994).
	Averaging Time - Noncancer (days)	10950	exposure period * 365 days/year.
	Averaging Time - Lifetime (days)	25,550	70-yr lifetime exposure period * 365 days/year.
Trespasser/Recreational Receptor	**Incidental Ingestion of & Dermal Contact with Sediment**		
	Ingestion Rate (mg/day)	50	adult soil ingestion rate (U.S. EPA, 1991).
	Fraction from Site (unitless)	1	assume, on days exposed, all soil exposure occurs at the site.
	Surface Area Exposed (cm^2/day)	19,400	average adult total body surface area (U.S. EPA, 1990; IRP, 1994).
	Soil-to-Skin Adherence Factor (mg/cm^2)	1	upper bound value (U.S. EPA, 1992; IRP, 1994).
	Exposure Frequency (days/yr)	7	IRP, 1994.
	Exposure Duration (yr)	30	Default residential exposure duration (U.S. EPA, 1991; IRP, 1994).
	Body Weight (kg)	70	average adult body weight (EPA, 1990; IRP, 1994).
	Averaging Time - Noncancer (days)	10,950	exposure period * 365 days/year.
	Averaging Time - Lifetime (days)	25,550	70-yr lifetime exposure period * 365 days/year.

Notes:

IRP, 1994. Final Risk Assessment Handbook, Massachusetts Military Reservation, Cape Cod, Massachusetts. *September.*

MADEP. 1994. Background Documentation for the Development of the MCP Numerical Standards. Bureau of Waste Site Cleanup and Office of Research and Standards. April 1994.

MADEP. 1995. Guidance for Disposal Site Risk Characterization - In Support of the Massachusetts Contingency Plan. Interim Final Policy. WSC/ORS-95-141.

U.S. EPA. 1989. Risk Assessment Guidance for Superfund: Volume I Human Health Evaluation Manual. Part A. Interim Final. EPA/540/1-89/002.

U.S. EPA. 1990. Exposure Factors Handbook. EPA/600/8-89/043.

U.S. EPA. 1991. Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors." OSWER Directive 9285.6-03.

U.S. EPA. 1992. Dermal Exposure Assessment: Principles and Applications. Interim Report. EPA/600/8-91/011B.

used to estimated potential health risk (EPA Region I, 1995). For surface water and sediment, UCLs will be calculated for individuals ponds that are capable of supporting recreational activities.

According to the Action Plan (ETA, 1997), shallow soil samples will be collected at gun and mortar positions and other potential source areas within the Impact Area from 0 - 0.5 ft. below ground surface (bgs) and 0.5 - 2 ft. bgs to determine whether munitions have impacted soil quality. Additional samples may be collected from 2 - 4 ft. bgs, depending on the results of the shallow samples. Subsurface soil samples will be collected at 10 ft. intervals and the top and bottom of the unsaturated zone (ETA, 1997). Additional deep samples may be collected, as needed, based on the data generated from the current work scope.

Soil EPCs will be generated using data collected from the 0 - 2 ft. horizon, consistent with the exposure scenarios for the Study Area, which include only potential surface exposures. Use of this dataset is also consistent with EPA Region I guidance (1995), which defines surface soil as 0 - 1 ft. bgs. The lower depth was set at 2 ft. bgs., rather than 1 ft. bgs., because of the manner in which soil samples will be collected (i.e., composites from the 0.5 to 2 ft. interval) and thus, there will not be any samples collected to a lower depth of 1 ft. bgs. Soil data generated from samples collected greater than 2 ft. bgs will not be included in calculation of EPCs. Note, however, that the potential for constituents detected in soil from any depth to leach to groundwater is specifically addressed in *the Draft Response Matrix for the Camp Edwards Impact Area Groundwater Quality Study*, prepared by Ogden (1997).

Soil EPCs will be calculated as described above for each exposure area. At this time, the following exposure areas have been identified:

- Impact Area: Because trespassers/recreational users are the most exposed receptor in this area and their movement cannot be determined, it will be assumed that there is equal likelihood of contacting soil in any location within the Impact Area.
- Individual Shooting Ranges: ARNG and law enforcement personnel undertake small arms training at individual ranges within the Training Range Area. It is not likely that personnel shoot at one target and then move to another. Therefore, each range will be considered a separate exposure area.

3.3.5 Average Daily Dose Calculations

The Average Daily Dose (ADD) is calculated to estimate a receptor's potential daily intake from exposure to compounds detected in environmental media. According to U.S. EPA (1989) and MADEP (1995), for compounds with potential noncarcinogenic effects, the exposure dose should be calculated by averaging over the period of time for which the receptor is assumed to be exposed. For compounds with potential carcinogenic effects, the lifetime ADD is calculated to estimate potential exposures over the course of a lifetime.

The ADD equations that will be used to evaluate potential exposures in this risk assessment are consistent with equations presented by U.S. EPA (1989, 1990, 1991, 1992) and MADEP (1995), and are shown below:

3.3.5.1 Ingestion of Groundwater

$$ADD = \frac{CW \times IR \times FI \times RAFo \times EF \times ED}{BW \times AT}$$

where:

ADD = Average Daily Dose Due to Ingestion (mg/kg-day)
CW = Compound Concentration in Water (mg/L)
IR = Water Ingestion Rate (L/day)
FI = Fraction of Total Intake From Study Area (unitless)
RAFo = Relative Absorption Factor (Oral-Water) (unitless)
EF = Exposure Frequency (days/year)
ED = Exposure Duration (years)
BW = Body Weight (kg)
AT = Averaging Time (ED x 365 days/yr, noncancer; 70yr x 365 days/yr, cancer)

3.3.5.2 Incidental Ingestion of Soil or Sediment

$$ADD = \frac{CS \times IR \times FI \times RAFo \times EF \times ED \times CF}{BW \times AT}$$

where:

ADD =	Average Daily Dose Due to Ingestion (mg/kg-day)
CS =	Compound Concentration in Soil (mg/kg)
IR =	Soil Ingestion Rate (mg/day)
FI =	Fraction of Soil Ingested From the Site (unitless)
RAFo =	Relative Absorption Factor (Oral-Soil) (unitless)
EF =	Exposure Frequency (days/year)
ED =	Exposure Duration (years)
CF =	Conversion Factor (10^{-6} kg/mg)
BW =	Body Weight (kg)
AT =	Averaging Time (ED x 365 days/yr, noncancer; 70yr x 365 days/yr, cancer)

Note that while the equation for estimating exposure to soil or sediment is the same, individual exposure parameters, particularly exposure frequency, may differ between the estimate for soil contact *versus* sediment contact.

3.3.5.3 Dermal Contact with Soil or Sediment

$$ADD = \frac{CS \times SA \times AF \times RAFd \times EF \times ED \times CF}{BW \times AT}$$

where:

ADD =	Average Daily Dose Due to Dermal Contact (mg/kg-day)
CS =	Compound Concentration in Soil (mg/kg)
SA =	Skin Surface Area Exposed (cm^2/day)
AF =	Soil to Skin Adherence Factor (mg/cm^2)
RAFd =	Relative Absorption Factor (Dermal-Soil) (unitless)
EF =	Exposure Frequency (days/year)
ED =	Exposure Duration (years)
CF =	Conversion Factor (10^{-6} kg/mg)
BW =	Body Weight (kg)
AT =	Averaging Time (ED x 365 days/yr, noncancer; 70yr x 365 days/yr, cancer)

Note that while the equation for estimating exposure to soil or sediment is the same, individual exposure parameters, particularly exposure frequency, may differ between the estimate for soil contact *versus* sediment contact.

3.3.5.4 Inhalation of Soil-Derived Dust

$$ADD = \frac{CS \times (1/PEF) \times IR \times ET \times EF \times ED}{BW \times AT}$$

where:

ADD =	Average Daily Dose Due to Inhalation (mg/kg-day)
CS =	Compound Concentration in Soil (mg/kg)
PEF =	Particulate Emission Factor (m^3/kg)
IR =	Inhalation Rate (m^3/hr)
ET =	Exposure Time (hr/day)
EF =	Exposure Frequency (days/year)
ED =	Exposure Duration (years)
BW =	Body Weight (kg)
AT =	Averaging Time (ED x 365 days/yr, noncancer; 70yr x 365 days/yr, cancer)

3.3.5.5 Incidental Ingestion of Surface Water

$$ADD = \frac{CW \times IR \times FI \times RAFo \times ET \times EF \times ED \times CF}{BW \times AT}$$

where:

ADD =	Average Daily Dose Due to Ingestion (mg/kg-day)
CW =	Compound Concentration in Water (mg/L)
IR =	Incidental Water Ingestion Rate (ml/hr)
RAFo =	Relative Absorption Factor (Oral-Water) (unitless)
ET =	Exposure Time (hr/day)
EF =	Exposure Frequency (days/year)
ED =	Exposure Duration (years)
CF =	Conversion Factor (10^{-3} L/ml)
BW =	Body Weight (kg)
AT =	Averaging Time (ED x 365 days/yr, noncancer; 70yr x 365 days/yr, cancer)

3.3.5.6 Dermal Contact with Surface Water

$$ADD = \frac{CW \times SA \times DA \times RAFd \times EF \times ED \times CF}{BW \times AT}$$

where:

ADD =	Average Daily Dose Due to Dermal Contact (mg/kg-day)
CW =	Compound Concentration in Water (mg/L)
SA =	Skin Surface Area Exposed (cm^2/event)
DA =	The Unit Absorbed Dose (cm/event)
RAFd =	Relative Absorption Factor (Dermal-Water) (unitless)
EF =	Exposure Frequency (events/year)
ED =	Exposure Duration (years)
CF =	Conversion Factor (10^{-3} L/cm^3)
BW =	Body Weight (kg)
AT =	Averaging Time (ED x 365 days/yr, noncancer; 70yr x 365 days/yr, cancer)

Calculation of the Permeability Constant and Unit Absorbed Dose (DA)

U.S. EPA Guidance (1992) recommends a method for calculating uptake using a permeability constant, K_p. The permeability constant typically has units of length per time (e.g., cm/hour) and represents the movement of compounds across the skin at steady state. U.S. EPA provides K_p values where empirical measurements are available, as well as recommendations for estimating K_p for other compounds. The recommendation is that Kp for inorganic compounds be set at 0.001 cm/hour where no empirical value is available, and Kp for organic compounds be determined as a function of oil water partition coefficient (K_{ow}) and molecular weight (MW), using the following formula:

$$Log\, K_p = -2.72 + 0.71\, K_{ow} - 0.0061\, MW$$

These recommendations will be used for the exposure assessment in this study.

Under the assumption of steady-state absorption, the absorbed dose of a compound may be calculated using the formula:

$$DA_{event} = K_p\; CW\; ET$$

where:

DA_{event}= dose absorbed per skin area exposed per exposure event (mg/cm^2 event)
K_p = permeability coefficient (cm/hour)
CW = chemical concentration in water (mg/cm^3)
ET = duration of the exposure event (hour/event)

While steady state absorption is reasonable for inorganic compounds, organic chemicals are most likely passing through the skin by dissolving in the lipid membrane of skin and then partitioning to the blood stream. The ramifications of this mechanism are that (1) there is a "lag time" before steady state absorption ensues and that exposure may continue after the receptor is no longer in contact with water, due to the continued partitioning of compound

stored in skin lipids. To account for this "non-steady state" uptake, U.S. EPA (1992) provides a series of equations for calculating DA_{event}, that differ depending on the relationship of the lag time to the assumed duration of the exposure event. These equations will also be used for the exposure assessment in this study.

In the formula in 3.3.5.6, Ogden uses a DA_{event} value that does not incorporate chemical concentrations, since they are unknown at this time (CW is a separate parameter in the equation). This may be viewed as a "unit DA", i.e., an absorbed dose per unit of water concentration, and has the units of cm per event.

Table 6 provides unit DA_{event} calculations for the constituents of primary focus for the Training Range and Impact Area, under the assumption that the swimming event lasts 2.6 hours, as specified in the RAH (IRP, 1994). It can be seen that Kp values were only available for mercury and zinc, forcing the uses of default assumptions or calculations for all other constituents.

3.3.6 Lead Exposure

As previously mentioned, the evaluation of lead exposures is somewhat different than other constituents, because the exposure estimate must be compared to a blood lead level. U.S. EPA has developed a model (the Integrated Exposure and Uptake Biokinetic Model ; IEUBK) capable of computing blood lead levels in children. This model will be used to evaluate present and future risks associated with lead in drinking water, as necessary.

The IEUBK model does not predict blood lead levels in adults. This is of no consequence for drinking water exposures, as it would be anticipated that exposure in children would represent the highest exposure and therefore, control risk management decisions. However, the exposure assessment contemplates present and future soil exposures that are limited to adults. To evaluate these exposures U.S. EPA (1996) has recently recommended a method for determining an upper bound on fetal blood lead level, $PbB_{fetal\ 0.95}$, that may occur as the result of exposure to soil lead in women of child bearing age. This model was developed by EPA for use in an industrial/commercial setting and has been applied by EPA to other small arms firing ranges.

Table 6
Parameters for Calculating Dermal Uptake from Water
Human Health Risk Assessment
Impact Area, Massachusetts Military Reservation

Compound	Kow (l/kg)	MW	Physical Property Reference	Kp (cm/hr)	Kp Reference	Unit Absorbed Dose (Da) cm/event
EXPLOSIVES						
HMX	6.6E-06	296	Howard and Meylan, 1997	6.26E-09	Calculated	6.59E-08
RDX	0.9	222	HSDB	7.64E-05	Calculated	4.79E-04
1,3-Dinitrobenzene	1.5	168	MMR Handbook (IRP, 1994)	2.39E-04	Calculated	1.06E-03
1,3,5-Trinitrobenzene	1.2	213	MMR Handbook (IRP, 1994)	1.08E-04	Calculated	6.33E-04
Tetryl	1.7	287	MMR Handbook (IRP, 1994)	4.83E-05	Calculated	4.78E-04
Nitrobenzene	1.9	123	MMR Handbook (IRP, 1994)	5.24E-04	Calculated	1.88E-03
2,4,6-Trinitrotoluene	1.6	227	HSDB	1.10E-04	Calculated	7.12E-04
2-Amino-4,6-Dinitrotoluene	1.9	197	assumed to be equal to isomer listed below	1.88E-04	Calculated	9.89E-04
4-Amino-2,6-Dinitrotoluene	1.9	197	MMR Handbook (IRP, 1994)	1.88E-04	Calculated	9.89E-04
2,6-Dinitrotoluene	1.5	182	MMR Handbook (IRP, 1994)	2.01E-04	Calculated	9.51E-04
2,4-Dinitrotoluene	2.0	182	MMR Handbook (IRP, 1994)	2.43E-04	Calculated	1.15E-03
2-Nitrotoluene	2.3	137	HSDB	5.02E-04	Calculated	1.91E-03
3-Nitrotoluene	2.5	137	HSDB	5.26E-04	Calculated	2.00E-03
4-Nitrotoluene	2.4	137	HSDB	5.13E-04	Calculated	1.95E-03
2,6-Diamino-4-nitrotoluene	2.5	171	3-Nitrotoluene surrogate	3.31E-04	Calculated	1.50E-03
2,4-Diamino-6-nitrotoluene	2.5	171	3-Nitrotoluene surrogate	3.31E-04	Calculated	1.50E-03
PETN	0.6	316	MMR Handbook (IRP, 1994)	1.55E-05	Calculated	1.88E-04
Picric Acid	21.4	229	Howard and Meylan, 1997	6.71E-04	Calculated	4.42E-03
Ammonium Picrate	0.04	246	Howard and Meylan, 1997	6.09E-06	Calculated	4.52E-05
INORGANICS						
Antimony				0.001	assumed (EPA, 1992)	0.0026
Arsenic				0.001	assumed (EPA, 1992)	0.0026
Barium				0.001	assumed (EPA, 1992)	0.0026
Copper				0.001	assumed (EPA, 1992)	0.0026
Lead				0.001	EPA, 1992	0.0026
Mercury (inorganic)				0.001	EPA, 1992	0.0026
Zinc				0.0006	EPA, 1992	0.00156

The adult lead model may be reduced to the following equation:

$$\mathrm{PbB}_{\mathrm{fetal},0.95} = \left[\mathrm{PbB}_{\mathrm{adult},0} + \frac{PbS * BKSF * IR_s * AF_s * EF}{AT}\right] * GSD_{adult}^{1.645} * R_{fetal/maternal}$$

where:

$\mathrm{PbB}_{\mathrm{fetal}\ 0.95}$ = the upper 95 percentile on blood lead levels in fetuses of mothers exposed to soil lead (ug/dL)

$\mathrm{PbB}_{\mathrm{adult},0}$ = blood lead levels in adults from background sources (ug/dL)

BKSF = uptake factor relating lead intake to blood lead (unitless)

IR = soil ingestion rate (mg/day)

AF = absorption fraction of lead in soil (unitless)

EF = exposure frequency (days/year)

AT = averaging time (365 days/year)

GSD = geometric standard deviation of blood lead levels in women of child bearing age, as determined from the literature or site-specific information on variation in blood lead levels among receptors.

$R_{\mathrm{fetal/maternal}}$ = the ratio of fetal to maternal blood lead level.

The ingestion rate and exposure frequency that will be applied to this equation are presented in Table 5. Other parameter values will be the defaults recommended in U.S. EPA (1996) unless newer or site-specific information is available at the time of the exposure assessment.

3.4 Risk Characterization

Potential noncarcinogenic and carcinogenic risks will be estimated for all identified receptors.

3.4.1 Noncancer Risk

The potential noncarcinogenic risk, or Hazard Quotient, is calculated as follows:

$$HQ = \frac{ADD}{RfD}$$

where:

HQ = Hazard Quotient (unitless);
ADD = Average Daily Dose (mg/kg-day); and
RfD = Reference Dose (mg/kg-day).

The HQ for all COPCs and exposure pathways will be summed to estimate the cumulative noncancer risk, or Hazard Index, for each receptor. The HIs calculated for each receptor will be compared to EPA's and MADEP's noncancer risk limit of 1.0. Should an HI for a receptor exceed the noncancer risk limit, then endpoint-specific HIs will be calculated and compared to the noncancer risk limit.

3.4.2 Cancer Risk

Potential carcinogenic risk, or Excess Lifetime Cancer Risk (ELCR), is calculated as follows:

$$ELCR = CSF \times LADD$$

where:

ELCR = Excess Lifetime Cancer Risk (unitless)
CSF = Cancer Slope Factor (1/(mg/kg-day)); and
LADD = Lifetime Average Daily Dose (mg/kg-day).

Pathway-specific ELCR estimates will be calculated by summing the ELCR for individual COPCs, and, cumulative site ELCR estimates will be calculated for each receptor by summing the pathway-specific ELCRs. The cumulative site ELCR for each receptor will be compared to a ELCR limit of 1×10^{-5} (one in one hundred thousand) This cancer risk limit was selected because it is MADEP's Cumulative Cancer Risk Limit, and because it is within EPA's acceptable risk range of 1×10^{-6} to 1×10^{-4} (one in one million to one in one hundred thousand).

3.4.3 Risk Due to Lead

Risks from exposure to lead in drinking water will be evaluated based on the outcome of the IEUBK model versus the goal of 95% of blood lead levels in children being below the 10 ug/dL concentration.

Risks from soil lead will be evaluated based on the outcome of the adult soil lead exposure model a goal of 95% of blood lead levels in potential fetuses of women of child bearing age being below the 10 ug/dL concentration.

3.5 Uncertainty Analysis

Each step of the risk assessment process involves making assumptions, due to the lack of absolute knowledge. These assumptions introduce some degree of uncertainty into the risk assessment. Thus, an uncertainty analysis is included in the risk assessment process to discuss those assumptions that impart the greatest uncertainty. General topics that will be discussed include: analytical data, COPC selection, toxicological data, and exposure assumptions.

4. REFERENCES

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APPENDIX A: PROPOSED PRELIMINARY RISK EVALUATION METHODOLOGY

A-1. INTRODUCTION

The Action Plan (ETA, 1997) states that a Preliminary Risk Evaluation (PRE) will be conducted in accordance with the MMR Risk Assessment Handbook (IRP, 1994). This approach is proposed for use in two aspects of risk assessment for the Impact Area Groundwater Quality Study: (1) the use of conservative risk-based concentrations to screen for chemical constituents of potential concern (COPCs) that should be evaluated in a quantitative risk assessment (Tier I) and (2) the use of a separate (Tier II) set of risk-based values to determine if any immediate, interim action should be taken to prevent human exposures to high chemical concentrations during the course of study and selection of final remedies, if any. The proposed Tier I and II concentrations are shown in Table A-1. The proposed approach to development of risk-based concentrations for application of the two-tiered PRE is discussed in the following sections.

The guidance in the Risk Assessment Handbook (RAH) has been supplemented with information from other sources including:

(1) EPA Integrated Risk Information Service (IRIS) database. Toxicity factors (Reference Doses and Cancer Slope Factors) were checked in IRIS and updated as required.

(2) EPA memos on risk assessment of other impact areas. Toxicity factors for certain explosives and propellants were not available in either IRIS or the RAH and were obtained from EPA sources who have previously considered the toxicity of such compounds.

(3) Guidance and support documents developed for the Massachusetts Contingency Plan. The RAH is currently being rewritten. It is Ogden's understanding that the revised RAH will do more to incorporate risk assessment procedures provided in guidance supporting both the Massachusetts Contingency Plan (MCP) and CERCLA actions (William Sweet, Brooks Air Force Base, May 15, 1997). Thus, Ogden also consulted MCP Guidance. The most important aspect of Ogden's proposal is that MCP Reportable Concentrations be used for Tier I screening, where they are available. This is discussed in Section 2.

(4) Professional Judgment. The existing RAH was developed for evaluation of discrete release areas and Areas of Concern identified during investigation of MMR, exclusive of the Impact Area. The exposure scenarios used to develop the Tier I and II values

presented in the RAH are dependent upon the location of the particular study area, (i.e., with the Flightline Area or outside the Security Zone) (IRP, 1994). However, the uses of and potential exposures within the Impact Area were not specifically considered in the development of the RAH because the Impact Area had not been the subject of investigative activity prior to the February 1997 Administrative Order.

Therefore, the PRE screening concentrations presented in the RAH may not be applicable to the Impact Area. For example, Tier I and Tier II soil screening values for outside the Security Zone are based on future residential use and current recreational use, respectively. Similarly, Tier I and II soil concentrations for inside the Flightline Area are based on current and future utility worker exposure. None of these scenarios is applicable to the Impact Area, which consists of undeveloped land used for ANG and law enforcement training purposes. As such, different exposure scenarios were applied herein to develop Tier I and II screening concentrations applicable to the Impact Area. As previously mentioned, these included the exposure underlying MCP Reportable Concentrations for Tier I and site-specific exposure assumptions for Tier II. Although site-specific exposure scenarios were developed, many of the exposure values (e.g., intake rates, exposure frequencies) used in these assumptions were obtained from the RAH (IRP, 1994).

The target risk levels and implementation of the PRE are as specified in the RAH. Tier I screening values represent health-based media concentrations assuming worst-case future scenarios and using a target cancer risk level of 1×10^{-6} and a target hazard quotient of 0.2. Tier II values represent health-based media concentrations assuming realistic current scenarios and using target risk levels of 1×10^{-3} and 10, respectively. According to the RAH, maximum detected media concentrations should be compared to the Tier I and II screening values. Exceedance of the Tier I screening values indicates that further evaluation is necessary, whereas exceedance of the Tier II screening values indicates that immediate response is warranted (e.g., fencing to restrict access). As implemented herein, the results of the PRE will be used to identify COPC and to identify potential imminent hazards. That is, constituents with maximum concentrations above the Tier I screening values will be considered COPCs and will be evaluated in a quantitative risk assessment. Exceedance of the Tier II screening values will trigger evaluation of whether interim measures are required to minimize any immediate potential hazard.

A.2. PROPOSED TIER I SCREENING CONCENTRATIONS

Tier I screening concentrations represent acceptable media concentrations based on the worst-case potential future site use and are intended to be compared with maximum detected

concentrations in a study area (IRP, 1994). Thus, exceedances of the Tier I concentrations indicate only that further evaluation is required.

A.2.1 Groundwater and Soil Tier I Values

The purpose of Tier I screening values for groundwater and soil, as described in the RAH (IRP, 1994), is similar to that of MADEP's Reportable Concentrations (RCs) in groundwater and soil. As described in 310 CMR 40.0630 of the MCP, RCs are screening concentrations that indicate a release has occurred and require notification to MADEP. Groundwater and soil RCs represent the lowest of the MCP Method 1 groundwater and soil standards , respectively (MADEP, 1994). The groundwater RCs are protective of consumption, indoor air, and surface water discharge scenarios. The soil RCs are protective of direct contact and, for organic compounds, leaching to groundwater is also included. The target risk levels used in the MCP are identical to those specified in the RAH.

Therefore, Ogden proposes the use of Category 1 groundwater and soil RCs as the Tier I screening concentrations for the Impact Area PRE. Reportable Concentration categories RCGW-1 and RCS-1, which apply to situations with high exposure potential (MADEP, 1994), will be used. The use of these values is justified because they are for the most part based on residential exposure and thus, conservative for use at the Impact Area.

Where Reportable Concentrations for groundwater and soil are not available, the algorithms developed by the MADEP for calculating Reportable Concentrations will be used to develop Tier I values. This was the case for several metals and the explosives and propellants listed in Table 1 of this Risk Assessment Work Plan[1]. Attachment 1 to this Appendix provides the algorithms and spreadsheet computations of groundwater and soil Tier I screening values for these compounds. Tier I screening concentrations will be calculated according to these equations for other constituents, if they are detected.

It should be noted that this calculation addresses the direct contact scenarios only. As previously mentioned, the MADEP Reportable Concentration methodology includes leaching potential for organic compounds only (RCs for inorganics are based on direct contact only). Further, for the explosive and propellants, it was not possible to calculate a leaching value, as these compounds do not appear to behave according to the typical paradigm applied to organic compounds (leaching as a function of organic carbon partition coefficient, K_{oc}). This means that, in contrast

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[1] In actuality, Reportable Concentrations are available for several of the explosives. However, these were not developed using the typical risk-based system. Rather, they are default concentrations based on the Massachusetts Reportable Quantity. It was felt that it would be more consistent with the RAH to compute risk-based concentrations for these compounds.

to most organic compounds, Tier I values for metals, explosives and propellants cannot be used a the sole criteria for determining if a quantitative risk assessment of these compounds is necessary.

However, as noted in the *Draft Response Matrix for the Camp Edwards Impact Area Groundwater Quality Study* (Ogden, 1997a), an evaluation of the potential for leaching of a compound to groundwater is triggered by the finding of any detectable concentration of the compounds of primary interest (as noted in Table 1 of the Risk Assessment Work Plan) in soil. Thus, these studies provide a supplement to the Tier I screening of soil. In the event that such studies are required, one part of the evaluation will be to estimate the potential groundwater concentration produced by compounds detected in soil. This estimated concentration will be used in Tier I groundwater screening. And, if screening values are exceeded, as an exposure point concentration for quantitative risk assessment.

A.2.2 Tier I Values for Surface Water and Sediment

MADEP has not developed RCs for surface water and sediment. Therefore, Ogden proposes to calculate Tier I surface water and sediment screening concentrations using the exposure assumptions and equations presented in the RAH, with the exception that inhalation exposures will not be considered. Inhalation exposures are not considered relevant to the explosives and metals identified as the primary constituents of concern, due to their low or non-volatility. Should volatile constituents be detected in surface water or sediment, inclusion of this pathway will be considered.

Attachment 2 presents equations and spreadsheet calculation of surface water and sediment Tier I screening concentrations for the explosives and metals that are the primary constituents of interest at the Impact Area. These calculations utilize a rearrangement of the exposure equations presented in Sections 3.3.5 of this Risk Assessment Work Plan to solve for a concentration achieving the target risk levels specified in the RAH (IRP, 1994). The exposure assumptions for the trespasser/recreational receptor, as provided in Table 3 of the Risk Assessment Work Plan were used. Tier I screening concentrations will be calculated according to these equations for other constituents, if they are detected.

A.3. PROPOSED TIER II SCREENING CONCENTRATIONS

Tier II screening concentrations represent acceptable media concentrations based on more realistic potential current exposures and are intended to be compared with maximum detected concentrations (IRP, 1994). In contrast to the Tier I concentrations, Tier II concentrations

presented in the RAH correspond to a risk level of 1×10^{-3} (one in 1,000) and a hazard quotient of 10. Ogden will use Tier II screening concentrations to determine if an interim response to prevent an imminent hazard is required.

Ogden proposes to develop Tier II screening concentrations by combining and rearranging the site-specific exposure equations presented in this Risk Assessment Work Plan to solve for concentrations that would be protective at the higher Tier II target risk levels. Tier II groundwater concentrations will be calculated based on ingestion exposure only (see Section 3.3.2 of the Risk Assessment Work Plan) and thus, the equation presented in Section 3.3.5.1 of the Risk Assessment Work Plan will simply be rearranged to solve for concentration. For soil, the equations used to calculate potential exposure dose via ingestion, dermal contact, and inhalation of soil-derived dust will be combined and rearranged to solve for a concentration that is protective of all three potential soil exposures. The equations to calculate potential surface water and sediment exposures via ingestion and dermal contact will be similarly combined and rearranged to solve for surface water and sediment concentrations protective of these potential exposures.

As noted in Table 2 of the Risk Assessment Work Plan, more than one receptor may be potentially exposed to soil. The exposure parameters for the ARNG Personnel receptor, provided in Table 3 of the Risk Assessment Work Plan, were used to derive Tier II soil screening values for the Training Range Area; and, the exposure parameters for the Trespasser/Recreational Receptor, also provided in Table 3 of the Risk Assessment Work Plan, were used to derive Tier II soil screening values for the Impact Area. These receptors are anticipated to be the most exposed site-specific receptors for each Area, based on the parameters provided.

Attachment 3 presents the equations and spreadsheet calculations of Tier II screening concentrations for the primary constituents of interest. Tier II screening concentrations will be calculated for other constituents as they are detected, according to these equations.

Table A-1
Tier I and II Screening Concentrations
Human Health Risk Assessment
Impact Area, Massachusetts Military Reservation

	SOIL						GROUNDWATER				SURFACE WATER				SEDIMENT			
	Tier I (mg/kg)	Basis	Tier II - Training Range (mg/kg)	Basis	Tier II - Impact Area (mg/kg)	Basis	Tier I (µg/l)	Basis	Tier II (µg/l)	Basis	Tier I (mg/l)	Basis	Tier II (mg/l)	Basis	Tier I (mg/kg)	Basis	Tier II (mg/kg)	Basis
EXPLOSIVES																		
HMX	576	Calculated[1]	176,938	Calculated[2]	280,153	Calculated[2]	350	Calculated[1]	17,500	Calculated[3]	281	Calculated[4]	14,038	Calculated[4]	3,744	Calculated[4]	187,179	Calculated[4]
RDX	2.24	Calculated[1]	9,008	Calculated[2]	11,885	Calculated[2]	0.32	Calculated[1]	318	Calculated[3]	0.56	Calculated[4]	556	Calculated[4]	7.94	Calculated[4]	7,941	Calculated[4]
1,3-Dinitrobenzene	1.15	Calculated[1]	354	Calculated[2]	560	Calculated[2]	0.70	Calculated[1]	35	Calculated[3]	0.48	Calculated[4]	24	Calculated[4]	7.49	Calculated[4]	374	Calculated[4]
1,3,5-Trinitrobenzene	0.58	Calculated[1]	177	Calculated[2]	280	Calculated[2]	0.35	Calculated[1]	18	Calculated[3]	0.26	Calculated[4]	13	Calculated[4]	3.74	Calculated[4]	187	Calculated[4]
Tetryl	576	Calculated[1]	176,938	Calculated[2]	280,153	Calculated[2]	350	Calculated[1]	17,500	Calculated[3]	262	Calculated[4]	13,105	Calculated[4]	3,744	Calculated[4]	187,179	Calculated[4]
Nitrobenzene	5.76	Calculated[1]	1,769	Calculated[2]	2,802	Calculated[2]	3.50	Calculated[1]	175	Calculated[3]	2.19	Calculated[4]	110	Calculated[4]	37	Calculated[4]	1,872	Calculated[4]
2,4,6-Trinitrotoluene	5.76	Calculated[1]	1,769	Calculated[2]	2,802	Calculated[2]	1.17	Calculated[1]	175	Calculated[3]	1.97	Calculated[4]	127	Calculated[4]	29	Calculated[4]	1,872	Calculated[4]
2-Amino-4,6-Dinitrotoluene	5.76	Calculated[1]	1,769	Calculated[2]	2,802	Calculated[2]	1.17	Calculated[1]	175	Calculated[3]	1.90	Calculated[4]	122	Calculated[4]	29	Calculated[4]	1,872	Calculated[4]
4-Amino-2,6-Dinitrotoluene	5.76	Calculated[1]	1,769	Calculated[2]	2,802	Calculated[2]	1.17	Calculated[1]	175	Calculated[3]	1.90	Calculated[4]	122	Calculated[4]	29	Calculated[4]	1,872	Calculated[4]
2,6-Dinitrotoluene	0.36	Calculated[1]	1,457	Calculated[2]	1,923	Calculated[2]	0.05	Calculated[1]	51	Calculated[3]	0.08	Calculated[4]	84	Calculated[4]	1.28	Calculated[4]	1,285	Calculated[4]
2,4-Dinitrotoluene	0.70	RCS-1	1,457	Calculated[2]	1,923	Calculated[2]	30	RCGW-1	51	Calculated[3]	0.08	Calculated[4]	82	Calculated[4]	1.28	Calculated[4]	1,285	Calculated[4]
2-Nitrotoluene	115	Calculated[1]	35,388	Calculated[2]	56,031	Calculated[2]	70	Calculated[1]	3,500	Calculated[3]	44	Calculated[4]	2,185	Calculated[4]	749	Calculated[4]	37,436	Calculated[4]
3-Nitrotoluene	115	Calculated[1]	35,388	Calculated[2]	56,031	Calculated[2]	70	Calculated[1]	3,500	Calculated[3]	43	Calculated[4]	2,163	Calculated[4]	749	Calculated[4]	37,436	Calculated[4]
4-Nitrotoluene	115	Calculated[1]	35,388	Calculated[2]	56,031	Calculated[2]	70	Calculated[1]	3,500	Calculated[3]	43	Calculated[4]	2,175	Calculated[4]	749	Calculated[4]	37,436	Calculated[4]
2,6-Diamino-4-Nitrotoluene	115	Calculated[1]	35,388	Calculated[2]	56,031	Calculated[2]	70	Calculated[1]	3,500	Calculated[3]	46	Calculated[4]	2,294	Calculated[4]	749	Calculated[4]	37,436	Calculated[4]
2,4-Diamino-6-Nitrotoluene	115	Calculated[1]	35,388	Calculated[2]	56,031	Calculated[2]	70	Calculated[1]	3,500	Calculated[3]	46	Calculated[4]	2,294	Calculated[4]	749	Calculated[4]	37,436	Calculated[4]
PETN	-		-				-		-		-		-		-		-	
Picric Acid	35	Calculated[1]	10,616	Calculated[2]	16,809	Calculated[2]	21	Calculated[1]	1,050	Calculated[3]	10	Calculated[4]	508	Calculated[4]	225	Calculated[4]	11,231	Calculated[4]
Ammonium Picrate	35	Calculated[1]	10,616	Calculated[2]	16,809	Calculated[2]	21	Calculated[1]	1,050	Calculated[3]	17	Calculated[4]	837	Calculated[4]	225	Calculated[4]	11,231	Calculated[4]
METALS																		
Antimony	10	RCS-1	53,773	Calculated[2]	85,156	Calculated[2]	6.0	RCGW-1	140	Calculated[3]	1.62	Calculated[4]	81	Calculated[4]	5,840	Calculated[4]	292,000	Calculated[4]
Arsenic	30	RCS-1	60,772	Calculated[2]	80,542	Calculated[2]	50	RCGW-1	23	Calculated[3]	0.03	Calculated[4]	31	Calculated[4]	277	Calculated[4]	276,965	Calculated[4]
Barium	4,516	Calculated[1]	---	Calculated[2]	---	Calculated[2]	490	Calculated[1]	24,500	Calculated[3]	283	Calculated[4]	14,160	Calculated[4]	---	Calculated[4]	---	Calculated[4]
Copper	2,396	Calculated[1]	---	Calculated[2]	---	Calculated[2]	260	Calculated[1]	13,000	Calculated[3]	150	Calculated[4]	7,513	Calculated[4]	542,286	Calculated[4]	---	Calculated[4]
Lead	300	RCS-1					20	RCGW-1									---	
Mercury	10	RCS-1	20,160	Calculated[2]	31,931	Calculated[2]	1.0	RCGW-1	53	Calculated[3]	0.23	Calculated[4]	12	Calculated[4]	2,190	Calculated[4]	109,500	Calculated[4]
Zinc	2,500	RCS-1	---	Calculated[2]	---	Calculated[2]	900	RCGW-1	65,625	Calculated[3]	731	Calculated[4]	36,537	Calculated[4]	---	Calculated[4]	---	Calculated[4]

Notes:

Tier I values are health-based screening concentrations calculated assuming a target risk level of $1x10^{-6}$ and a target hazard quotient of 0.2.

Tier II values are health-based screening concentrations calculated assuming a target risk level of $1x10^{-3}$ and a target hazard quotient of 10.

RCS-1 and RCGW-1 represent MCP Reportable Concentrations for soil and groundwater, respectively.

1. Tier I soil and groundwater were calculated using the equations and assumptions used by MADEP (1994) to calculate Category 1 Reportable Concentrations.
2. Tier II soil concentrations were calculated using the equations provided in the MMR Risk Assessment Handbook (IRP, 1994) and site-specific exposure assumptions for the Training Range and Impact Area, as described in the text.
3. Tier II groundwater concentrations were calculated using the equations and assumptions used for Tier I concentrations, substituting a target risk level of 10^{-3} and a target hazard quotient of 10.
4. Tier I and II surface water and sediment concentrations were calculated using the equations and assumptions provided in the MMR Risk Assessment Handbook (IRP, 1994).

---: This calculation resulted in a concentration greater than a part per part and therefore, this method is not applicable.

MADEP, 1994. *Background Documentation for the Development of the MCP Numerical Standards*, Massachusetts Department of Environmental Protection, April.

IRP, 1994. *Risk Assessment Handbook, Massachusetts Military Reservation, Cape Cod, Massachusetts*, Air National Guard Bureau, September.

Attachment 1

Calculation of Tier I Screening Concentrations for Groundwater and Soil

Tier I Risk-Based Screening Concentrations
Ingestion of Drinking Water

$$C_{dw} = \frac{0.2 \times RfD \times BW \times AP \times C}{IR \times RAF \times F \times D1 \times D2} \quad \text{(noncancer)}$$

$$C_{dw} = \frac{ELCR \times BW \times AP \times C}{IR \times RAF \times F \times D1 \times D2 \times CSF} \quad \text{(cancer)}$$

C_{dw} (µg/l)	Risk-based concentration in drinking water	calculated
0.2 (unitless)	20% Source Allocation Factor	
RfD (mg/kg/day)	Oral Reference Dose	see below
BW (kg)	Receptor's Body Weight	70
AP (yrs)	Averaging Period	70
C (µg/mg)	Units Conversion Factor	1E+3
IR (l/day)	Water Ingestion Rate	2
RAF (unitless)	Relative Absorption Factor	see below
F (event/day)	Frequency of Exposure	1
D1 (day/event)	Duration of Exposure Event	1
D2 (yrs)	Duration of Exposure Period	70
ELCR (unitless)	Target Excess Lifetime Cancer Risk	1E-6
CSF [(mg/kg/day)$^{-1}$]	Oral Cancer Slope Factor	see below

				C_{dw}	
COMPOUND	**RfD (mg/kg/day)**	**CSF [(mg/kg/day)-1]**	**RAF**	**noncancer (µg/l)**	**cancer (µg/l)**
EXPLOSIVES					
HMX	0.05	NA	1	350.00	NA
RDX	0.003	0.11	1	21.00	0.32
1,3-Dinitrobenzene	0.0001	NA	1	0.70	NA
1,3,5-Trinitrobenzene	0.00005	NA	1	0.35	NA
Tetryl	0.05	NA	1	350.00	NA
Nitrobenzene	0.0005	NA	1	3.50	NA
2,4,6-Trinitrotoluene	0.0005	0.03	1	3.50	1.17
2-Amino-4,6-Dinitrotoluene	0.0005	0.03	1	3.50	1.17
4-Amino-2,6-Dinitrotoluene	0.0005	0.03	1	3.50	1.17
2,6-Dinitrotoluene	0.001	0.68	1	7.00	0.05
2-Nitrotoluene	0.01	NA	1	70.00	NA
3-Nitrotoluene	0.01	NA	1	70.00	NA
4-Nitrotoluene	0.01	NA	1	70.00	NA
2,6-Diamino-4-Nitrotoluene	0.01	NA	1	70.00	NA
2,4-Diamino-6-Nitrotoluene	0.01	NA	1	70.00	NA
PETN			1		
Picric Acid	0.003	NA	1	21.00	NA
Ammonium Picrate	0.003	NA	1	21.00	NA
METALS					
Barium	0.07	NA	1	490.00	NA
Copper	0.037	NA	1	260.00	NA

Note:
Equations and inputs obtained from *Background Documentation for the Development of the MCP Numerical Standards,* Massachusetts Department of Environmental Protection (April 1994).

Tier 1 Risk-Based Screening Concentrations
Direct Contact Exposure with Contaminated Surface Soil

$$C_{soil} = \frac{0.2 \times RfD \times C}{(NADSIR \times RAF) + (NADSCR \times RAF)} \quad \text{(noncancer)}$$

$$C_{soil} = \frac{ELCR \times C}{((NLADSIR \times RAF) + (NLADSCR \times RAF)) \times CSF} \quad \text{(cancer)}$$

C_{soil} (mg/kg)	Risk-based concentration in soil	calculated
0.2 (unitless)	20% Source Allocation Factor	
RfD (mg/kg/day)	Oral Reference Dose	see below
C (mg/kg)	Units Conversion Factor	1E+6
NADSIR (mg_{soil}/kg/day)	Normalized Average Daily Soil Ingestion Rate	3.1
NADSCR (mg_{soil}/kg/day)	Normalized Average Daily Soil Dermal Contact Rate	28.5
RAF (unitless)	Relative Absorption Factor	see below
ELCR (unitless)	Target Excess Lifetime Cancer Risk	1E-6
NLADSIR (mg_{soil}/kg/day)	Time-weighted Normalized Lifetime Average Daily Soil Ingestion Rate	0.41
NLADSCR (mg_{soil}/kg/day)	Time-weighted Normalized Lifetime Average Daily Soil Dermal Contact Rate	7.3
CSF [$(mg/kg/day)^{-1}$]	Oral Cancer Slope Factor	see below

COMPOUND	RfD (mg/kg/day)	CSF [(mg/kg/day)-1]	RAF oral	RAF dermal	C_{soil} noncancer (mg/kg)	C_{soil} cancer (mg/kg)
EXPLOSIVES						
HMX	0.05	NA	1	0.5	576.37	NA
RDX	0.003	0.11	1	0.5	34.58	2.24
1,3-Dinitrobenzene	0.0001	NA	1	0.5	1.15	NA
1,3,5-Trinitrobenzene	0.00005	NA	1	0.5	0.58	NA
Tetryl	0.05	NA	1	0.5	576.37	NA
Nitrobenzene	0.0005	NA	1	0.5	5.76	NA
2,4,6-Trinitrotoluene	0.0005	0.03	1	0.5	5.76	8.21
2-Amino-4,6-Dinitrotoluene	0.0005	0.03	1	0.5	5.76	8.21
4-Amino-2,6-Dinitrotoluene	0.0005	0.03	1	0.5	5.76	8.21
2,6-Dinitrotoluene	0.001	0.68	1	0.5	11.53	0.36
2-Nitrotoluene	0.01	NA	1	0.5	115.27	NA
3-Nitrotoluene	0.01	NA	1	0.5	115.27	NA
4-Nitrotoluene	0.01	NA	1	0.5	115.27	NA
2,6-Diamino-4-Nitrotoluene	0.01	NA	1	0.5	115.27	NA
2,4-Diamino-6-Nitrotoluene	0.01	NA	1	0.5	115.27	NA
PETN			1	0.5		
Picric Acid	0.003	NA	1	0.5	34.58	NA
Ammonium Picrate	0.003	NA	1	0.5	34.58	NA
METALS						
Barium	0.07	NA	1	0	4516	NA
Copper	0.037	NA	1	0	2396	NA
Zinc	0.3	NA	1	0	19,354.84	NA

Note:
Equations and inputs obtained from *Background Documentation for the Development of the MCP Numerical Standards,* Massachusetts Department of Environmental Protection (April 1994).

Attachment 2

Calculation of Tier I Screening Concentrations for Surface Water and Sediment

Tier I Risk-Based Screening Concentrations
Ingestion and Dermal Contact with Surface Water

$$C_{sw} = \frac{0.2 \times RfD \times BW \times AT \times 365 \text{ days/yr}}{ED \times EF \times [(SA \times DA \times RAF \times CF) + (IR \times ET \times FI \times RAF)]} \quad \text{(noncancer)}$$

$$C_{sw} = \frac{ELCR \times BW \times AT \times 365 \text{ days/yr}}{ED \times EF \times [(SA \times DA \times RAF \times CF) + (IR \times ET \times FI \times RAF)] \times CSF} \quad \text{(cancer)}$$

C_{sw} (mg/l)	Risk-based concentration in surface water	calculated	
0.2 (unitless)	Target Hazard Quotient		
RfD (mg/kg/day)	Oral Reference Dose	see below	
BW (kg)	Receptor's Body Weight	70	
AT (yrs)	Averaging Time	30	noncancer
		70	cancer
ED (yrs)	Exposure Duration	30	
EF (days/yr)	Exposure Frequency	7	
SA (cm^2)	Skin Surface Area Available for Contact	19,400	
DA (cm)	Unit Absorbed Dose	see below	
RAF (unitless)	Relative Absorption Factor	see below	
CF (l/cm^3)	Volumetric Conversion Factor	0.001	
IR (l/hr)	Water Ingestion Rate	0.050	
ET (hr/day)	Exposure Time	2.6	
FI (unitless)	Fraction Ingested	1.0	
ELCR (unitless)	Target Excess Lifetime Cancer Risk	1E-6	
CSF [$(mg/kg/day)^{-1}$]	Oral Cancer Slope Factor	see below	

COMPOUND	RfD (mg/kg/day)	DA (cm)	RAF oral	RAF dermal	CSF [(mg/kg/day)-1]	C_{sw} noncancer (mg/l)	C_{sw} cancer (mg/l)
EXPLOSIVES							
HMX	0.05	6.59E-08	1	1	NA	280.77	NA
RDX	0.003	4.79E-04	1	1	0.11	15.72	0.56
1,3-Dinitrobenzene	0.0001	1.06E-03	1	1	NA	0.48	NA
1,3,5-Trinitrobenzene	0.00005	6.33E-04	1	1	NA	0.26	NA
Tetryl	0.05	4.78E-04	1	1	NA	262.09	NA
Nitrobenzene	0.0005	1.88E-03	1	1	NA	2.19	NA
2,4,6-Trinitrotoluene	0.0005	7.12E-04	1	1	0.03	2.54	1.97
2-Amino-4,6-Dinitrotoluene	0.0005	9.89E-04	1	1	0.03	2.45	1.90
4-Amino-2,6-Dinitrotoluene	0.0005	9.89E-04	1	1	0.03	2.45	1.90
2,6-Dinitrotoluene	0.001	9.51E-04	1	1	0.68	4.92	0.08
2,4-Dinitrotoluene	0.002	1.15E-03	1	1	0.68	9.59	0.08
2-Nitrotoluene	0.01	1.91E-03	1	1	NA	43.71	NA
3-Nitrotoluene	0.01	2.00E-03	1	1	NA	43.27	NA
4-Nitrotoluene	0.01	1.95E-03	1	1	NA	43.50	NA
2,6-Diamino-4-Nitrotoluene	0.01	1.50E-03	1	1	NA	45.89	NA
2,4-Diamino-6-Nitrotoluene	0.01	1.50E-03	1	1	NA	45.89	NA
PETN		1.88E-04	1	1			
Picric Acid	0.003	4.42E-03	1	1	NA	10.15	NA
Ammonium Picrate	0.003	4.52E-05	1	1	NA	16.73	NA
METALS							
Antimony	0.0004	2.60E-03	1	1	NA	1.62	NA
Arsenic	0.0003	2.60E-03	1	1	1.5	1.21	0.03
Barium	0.07	2.60E-03	1	1	NA	283.20	NA
Copper	0.037	2.60E-03	1	1	NA	150.27	NA
Mercury	0.0003	2.60E-03	2	13.7	NA	0.23	NA
Zinc	0.3	1.56E-03	1.6	3.03	NA	730.73	NA

Notes:
Equations and inputs were obtained from *Risk Assessment Handbook, Massachusetts Military Reservation, Cape Cod, Massachusetts*, Air National Guard Bureau (September 1994).
RAFs were added to these equations.

Tier 1 Risk-Based Screening Concentrations
Ingestion and Dermal Contact with Sediment

$$C_{sed} = \frac{0.2 \times RfD \times BW \times AT \times 365 \text{ days/yr}}{EF \times ED \times [(10^{-6} \text{ kg/mg} \times IR \times RAF) + (10^{-6} \text{ kg/mg} \times SA \times AF \times RAF)]} \quad \text{(noncancer)}$$

$$C_{sed} = \frac{ELCR \times BW \times AT \times 365 \text{ days/yr}}{[EF \times ED \times ((10^{-6} \text{ kg/mg} \times IR \times RAF) + (10^{-6} \text{ kg/mg} \times SA \times AF \times RAF))] \times CSF} \quad \text{(cancer)}$$

C_{sed} (mg/kg)	Risk-based concentration in sediment	calculated	
0.2 (unitless)	Target Hazard Quotient		
RfD (mg/kg/day)	Oral Reference Dose	see below	
BW (kg)	Receptor's Body Weight	70	
AT (yrs)	Averaging Time	30	noncancer
		70	cancer
EF (days/yr)	Exposure Frequency	7	
ED (yrs)	Exposure Duration	30	
IR (mg/day)	Sediment Ingestion Rate	50	
SA (cm^2/day)	Skin Surface Area Available for Contact	19,400	
RAF (unitless)	Relative Absorption Factor	see below	
AF (mg/cm^2)	Sediment to Skin Adherence Factor	1.0	
ELCR (unitless)	Target Excess Lifetime Cancer Risk	1E-6	
CSF [$(mg/kg/day)^{-1}$]	Oral Cancer Slope Factor	see below	

COMPOUND	RfD (mg/kg/day)	CSF [(mg/kg/day)-1]	RAF oral	RAF dermal	C_{sed} noncancer (mg/kg)	C_{sed} cancer (mg/kg)
EXPLOSIVES						
HMX	0.05	NA	1	0.5	3,744	NA
RDX	0.003	0.11	1	0.5	225	7.94
1,3-Dinitrobenzene	0.0001	NA	1	0.5	7.49	NA
1,3,5-Trinitrobenzene	0.00005	NA	1	0.5	3.74	NA
Tetryl	0.05	NA	1	0.5	3,744	NA
Nitrobenzene	0.0005	NA	1	0.5	37.44	NA
2,4,6-Trinitrotoluene	0.0005	0.03	1	0.5	37.44	29.12
2-Amino-4,6-Dinitrotoluene	0.0005	0.03	1	0.5	37.44	29.12
4-Amino-2,6-Dinitrotoluene	0.0005	0.03	1	0.5	37.44	29.12
2,6-Dinitrotoluene	0.001	0.68	1	0.5	74.87	1.28
2,4-Dinitrotoluene	0.002	0.68	1	0.5	150	1.28
2-Nitrotoluene	0.01	NA	1	0.5	749	NA
3-Nitrotoluene	0.01	NA	1	0.5	749	NA
4-Nitrotoluene	0.01	NA	1	0.5	749	NA
2,6-Diamino-4-Nitrotoluene	0.01	NA	1	0.5	749	NA
2,4-Diamino-6-Nitrotoluene	0.01	NA	1	0.5	749	NA
PETN			1	0.5		
Picric Acid	0.003	NA	1	0.5	225	NA
Ammonium Picrate	0.003	NA	1	0.5	225	NA
METALS						
Antimony	0.0004	NA	1	0	5,840	NA
Arsenic	0.0003	1.5	0.41	0	10,683	276.96
Barium	0.07	NA	1	0	1,022,000	NA
Copper	0.037	NA	1	0	542,286	NA
Mercury	0.0003	NA	2	0	2,190	NA
Zinc	0.3	NA	1	0	4,380,000	NA

Notes:
Equations and inputs were obtained from *Risk Assessment Handbook, Massachusetts Military Reservation, Cape Cod,* Massachusetts, Air National Guard Bureau (September 1994).

Attachment 3

Calculation of Tier II Screening Concentrations

Tier II Risk-Based Screening Concentrations
Ingestion of Drinking Water

$$C_{dw} = \frac{10 \times RfD \times BW \times AP \times C}{IR \times RAF \times F \times D1 \times D2} \quad \text{(noncancer)}$$

$$C_{dw} = \frac{ELCR \times BW \times AP \times C}{IR \times RAF \times F \times D1 \times D2 \times CSF} \quad \text{(cancer)}$$

C_{dw} (µg/l)	Risk-based concentration in drinking water	calculated
10 (unitless)	Target Hazard Quotient	
RfD (mg/kg/day)	Oral Reference Dose	see below
BW (kg)	Receptor's Body Weight	70
AP (yrs)	Averaging Period	70
C (µg/mg)	Units Conversion Factor	1E+3
IR (l/day)	Water Ingestion Rate	2
RAF (unitless)	Relative Absorption Factor	see below
F (event/day)	Frequency of Exposure	1
D1 (day/event)	Duration of Exposure Event	1
D2 (yrs)	Duration of Exposure Period	70
ELCR (unitless)	Target Excess Lifetime Cancer Risk	1E-3
CSF [(mg/kg/day)$^{-1}$]	Oral Cancer Slope Factor	see below

COMPOUND	RfD (mg/kg/day)	CSF [(mg/kg/day)-1]	RAF	C_{dw} noncancer (µg/l)	C_{dw} cancer (µg/l)
EXPLOSIVES					
HMX	0.05	NA	1	17,500	NA
RDX	0.003	0.11	1	1,050	318.18
1,3-Dinitrobenzene	0.0001	NA	1	35	NA
1,3,5-Trinitrobenzene	0.00005	NA	1	18	NA
Tetryl	0.05	NA	1	17,500	NA
Nitrobenzene	0.0005	NA	1	175	NA
2,4,6-Trinitrotoluene	0.0005	0.03	1	175	1166.67
2-Amino-4,6-Dinitrotoluene	0.0005	0.03	1	175	1166.67
4-Amino-2,6-Dinitrotoluene	0.0005	0.03	1	175	1166.67
2,6-Dinitrotoluene	0.001	0.68	1	350	51.47
2,4-Dinitrotoluene	0.002	0.68	1	700	51.47
2-Nitrotoluene	0.01	NA	1	3,500	NA
3-Nitrotoluene	0.01	NA	1	3,500	NA
4-Nitrotoluene	0.01	NA	1	3,500	NA
2,6-Diamino-4-Nitrotoluene	0.01	NA	1	3,500	NA
2,4-Diamino-6-Nitrotoluene	0.01	NA	1	3,500	NA
PETN			1		
Picric Acid	0.003	NA	1	1,050	NA
Ammonium Picrate	0.003	NA	1	1,050	NA
METALS					
Antimony	0.0004	NA	1	140	NA
Arsenic	0.0003	1.5	1	105	23.33
Barium	0.07	NA	1	24500	NA
Copper	0.037	NA	1	13000	NA
Mercury	0.0003	NA	2	53	NA
Zinc	0.3	NA	1.6	65,625	NA

Note:
Equations and inputs obtained from *Background Documentation for the Development of the MCP Numerical Standards,* Massachusetts Department of Environmental Protection (April 1994).

Tier II Risk-Based Screening Concentrations
Direct Contact Exposure with Contaminated Surface Soil - Impact Area

$$C_s = \frac{HI \times BW \times AT \times 365\ days/yr}{EF \times ED \times [(10^{-6}\ kg/mg \times (IR_{soil} \times RAF \times 1/RfD_o) + (SA \times AF \times RAF \times 1/RfD_o)) + (IR_{dust} \times ET \times 1/PEF \times 1/RfD_i)]} \quad \text{(noncancer)}$$

$$C_s = \frac{ELCR \times BW \times AT \times 365\ days/yr}{EF \times ED \times [(10^{-6}\ kg/mg \times (IR_{soil} \times RAF \times CSF_o) + (SA \times AF \times RAF \times CSF_o)) + (IR_{dust} \times ET \times 1/PEF \times CSF_i)]} \quad \text{(cancer)}$$

C_s (mg/kg)	Risk-based concentration in soil	calculated	
HI (unitless)	Target Hazard Index	10	
RfD_o (mg/kg/day)	Oral Reference Dose	see below	
BW (kg)	Receptor's Body Weight	70	
AT (yrs)	Averaging Time	30	noncancer
		70	cancer
EF (days/yr)	Exposure Frequency	24	
ED (yrs)	Exposure Duration	30	
IR_{soil} (mg/day)	Soil Ingestion Rate	50	
SA (cm^2/day)	Skin Surface Area Available for Contact	3,700	
RAF (unitless)	Relative Absorption Factor	see below	
AF (mg/cm^2)	Soil to Skin Adherence Factor	1.0	
RfD_i (mg/kg/day)	Inhalation Reference Dose	see below	
ELCR (unitless)	Target Excess Lifetime Cancer Risk	1E-3	
CSF_o [$(mg/kg/day)^{-1}$]	Oral Cancer Slope Factor	see below	
IR_{dust} (m^3/hr)	Inhalation Rate	2.0	
ET (hr/day)	Exposure Time	4	
PEF (m^3/kg)	Particulate Emission Factor	1.32E+09	
CSF_i [$(mg/kg/day)^{-1}$]	Inhalation Cancer Slope Factor	see below	

COMPOUND	RfD Oral (mg/kg/day)	RfD Inhalation (mg/kg/day)	CSF Oral [(mg/kg/day)-1]	CSF Inhalation [(mg/kg/day)-1]	RAF oral	RAF dermal	C_s noncancer (mg/kg)	C_s cancer (mg/kg)
EXPLOSIVES								
HMX	0.05	0.05	NA	NA	1	0.5	280,153	NA
RDX	0.003	0.003	0.11	0.11	1	0.5	16,809	11,885
1,3-Dinitrobenzene	0.0001	0.0001	NA	NA	1	0.5	560	NA
1,3,5-Trinitrobenzene	0.00005	0.00005	NA	NA	1	0.5	280	NA
Tetryl	0.05	0.05	NA	NA	1	0.5	280,153	NA
Nitrobenzene	0.0005	0.0006	NA	NA	1	0.5	2,802	NA
2,4,6-Trinitrotoluene	0.0005	0.0005	0.03	0.03	1	0.5	2,802	43,579
2-Amino-4,6-Dinitrotoluene	0.0005	0.0005	0.03	0.03	1	0.5	2,802	43,579
4-Amino-2,6-Dinitrotoluene	0.0005	0.0005	0.03	0.03	1	0.5	2,802	43,579
2,6-Dinitrotoluene	0.001	0.001	0.68	0.68	1	0.5	5,603	1,923
2,4-Dinitrotoluene	0.002	0.002	0.68	0.68	1	0.5	11,206	1,923
2-Nitrotoluene	0.01	0.01	NA	NA	1	0.5	56,031	NA
3-Nitrotoluene	0.01	0.01	NA	NA	1	0.5	56,031	NA
4-Nitrotoluene	0.01	0.01	NA	NA	1	0.5	56,031	NA
2,6-Diamino-4-Nitrotoluene	0.01	0.01	NA	NA	1	0.5	56,031	NA
2,4-Diamino-6-Nitrotoluene	0.01	0.01	NA	NA	1	0.5	56,031	NA
PETN					1	0.5		
Picric Acid	0.003	0.003	NA	NA	1	0.5	16,809	NA
Ammonium Picrate	0.003	0.003	NA	NA	1	0.5	16,809	NA
METALS								
Antimony	0.0004	0.0004	NA	NA	1	0	85,156	NA
Arsenic	0.0003	0.0003	1.5	15.1	0.41	0	155,747	80,542
Barium	0.07	0.0001	NA	NA	1	0	14,068,578	NA
Copper	0.037	0.037	NA	NA	1	0	7,907,375	NA
Mercury	0.0003	0.0001	NA	NA	2	0	31,931	NA
Zinc	0.3	0.3	NA	NA	1	0	63,867,259	NA

Notes:
Equations and inputs were obtained from *Risk Assessment Handbook, Massachusetts Military Reservation, Cape Cod, Massachusetts*, Air National Guard Bureau (September 1994).

Tier II Risk-Based Screening Concentrations
Direct Contact Exposure with Contaminated Surface Soil - Training Range Area

$$C_s = \frac{HI \times BW \times AT \times 365\ days/yr}{EF \times ED \times [(10^{-6}\ kg/mg \times ((IR_{soil} \times RAF \times 1/RfD_o) + (SA \times AF \times RAF \times 1/RfD_o)) + (IR_{dust} \times ET \times 1/PEF \times 1/RfD_i)]}\quad \text{(noncancer)}$$

$$C_s = \frac{ELCR \times BW \times AT \times 365\ days/yr}{EF \times ED \times [(10^{-6}\ kg/mg \times ((IR_{soil} \times RAF \times CSF_o) + (SA \times AF \times RAF \times CSF_o)) + (IR_{dust} \times ET \times 1/PEF \times CSF_i)]}\quad \text{(cancer)}$$

C_s (mg/kg)	Risk-based concentration in soil	calculated	
HI (unitless)	Target Hazard Index	10	
RfD_o (mg/kg/day)	Oral Reference Dose	see below	
BW (kg)	Receptor's Body Weight	70	
AT (yrs)	Averaging Time	25	noncancer
		70	cancer
EF (days/yr)	Exposure Frequency	38	
ED (yrs)	Exposure Duration	25	
IR_{soil} (mg/day)	Soil Ingestion Rate	50	
SA (cm^2/day)	Skin Surface Area Available for Contact	3,700	
RAF (unitless)	Relative Absorption Factor	see below	
AF (mg/cm^2)	Soil to Skin Adherence Factor	1.0	
RfD_i (mg/kg/day)	Inhalation Reference Dose	see below	
ELCR (unitless)	Target Excess Lifetime Cancer Risk	1E-3	
CSF_o [$(mg/kg/day)^{-1}$]	Oral Cancer Slope Factor	see below	
IR_{dust} (m^3/hr)	Inhalation Rate	2.5	
ET (hr/day)	Exposure Time	8	
PEF (m^3/kg)	Particulate Emission Factor	1.32E+09	
CSF_i [$(mg/kg/day)^{-1}$]	Inhalation Cancer Slope Factor	see below	

	RfD		CSF		RAF		C_s	
COMPOUND	Oral (mg/kg/day)	Inhalation (mg/kg/day)	Oral [(mg/kg/day)-1]	Inhalation [(mg/kg/day)-1]	oral	dermal	noncancer (mg/kg)	cancer (mg/kg)
EXPLOSIVES								
HMX	0.05	0.05	NA	NA	1	0.5	176,938	NA
RDX	0.003	0.003	0.11	0.11	1	0.5	10,616	9,008
1,3-Dinitrobenzene	0.0001	0.0001	NA	NA	1	0.5	354	NA
1,3,5-Trinitrobenzene	0.00005	0.00005	NA	NA	1	0.5	177	NA
Tetryl	0.05	0.05	NA	NA	1	0.5	176,938	NA
Nitrobenzene	0.0005	0.0006	NA	NA	1	0.5	1,769	NA
2,4,6-Trinitrotoluene	0.0005	0.0005	0.03	0.03	1	0.5	1,769	33,028
2-Amino-4,6-Dinitrotoluene	0.0005	0.0005	0.03	0.03	1	0.5	1,769	33,028
4-Amino-2,6-Dinitrotoluene	0.0005	0.0005	0.03	0.03	1	0.5	1,769	33,028
2,6-Dinitrotoluene	0.001	0.001	0.68	0.68	1	0.5	3,539	1,457
2,4-Dinitrotoluene	0.002	0.002	0.68	0.68	1	0.5	7,078	1,457
2-Nitrotoluene	0.01	0.01	NA	NA	1	0.5	35,388	NA
3-Nitrotoluene	0.01	0.01	NA	NA	1	0.5	35,388	NA
4-Nitrotoluene	0.01	0.01	NA	NA	1	0.5	35,388	NA
2,6-Diamino-4-Nitrotoluene	0.01	0.01	NA	NA	1	0.5	35,388	NA
2,4-Diamino-6-Nitrotoluene	0.01	0.01	NA	NA	1	0.5	35,388	NA
PETN					1	0.5		
Picric Acid	0.003	0.003	NA	NA	1	0.5	10,616	NA
Ammonium Picrate	0.003	0.003	NA	NA	1	0.5	10,616	NA
METALS								
Antimony	0.0004	0.0004	NA	NA	1	0	53,773	NA
Arsenic	0.0003	0.0003	1.5	15.1	0.41	0	98,323	60,772
Barium	0.07	0.0001	NA	NA	1	0	8,196,153	NA
Copper	0.037	0.037	NA	NA	1	0	4,993,224	NA
Mercury	0.0003	0.0001	NA	NA	2	0	20,160	NA
Zinc	0.3	0.3	NA	NA	1	0	40,329,884	NA

Notes:
Equations and inputs were obtained from *Risk Assessment Handbook, Massachusetts Military Reservation, Cape Cod,* Massachusetts, Air National Guard Bureau (September 1994).

Tier II Risk-Based Screening Concentrations
Ingestion and Dermal Contact with Surface Water

$$C_{sw} = \frac{10 \times RfD \times BW \times AT \times 365 \text{ days/yr}}{ED \times EF \times [(SA \times DA \times RAF \times CF) + (IR \times ET \times FI \times RAF)]} \quad \text{(noncancer)}$$

$$C_{sw} = \frac{ELCR \times BW \times AT \times 365 \text{ days/yr}}{ED \times EF \times [(SA \times DA \times RAF \times CF) + (IR \times ET \times FI \times RAF)] \times CSF} \quad \text{(cancer)}$$

C_{sw} (mg/l)	Risk-based concentration in surface water	calculated	
10 (unitless)	Target Hazard Quotient		
RfD (mg/kg/day)	Oral Reference Dose	see below	
BW (kg)	Receptor's Body Weight	70	
AT (yrs)	Averaging Time	30	noncancer
		70	cancer
ED (yrs)	Exposure Duration	30	
EF (days/yr)	Exposure Frequency	7	
SA (cm^2)	Skin Surface Area Available for Contact	19,400	
DA (cm)	Unit Absorbed Dose	see below	
RAF (unitless)	Relative Absorption Factor	see below	
CF (l/cm3)	Volumetric Conversion Factor	0.001	
IR (l/hr)	Water Ingestion Rate	0.050	
ET (hr/day)	Exposure Time	2.6	
FI (unitless)	Fraction Ingested	1.0	
ELCR (unitless)	Target Excess Lifetime Cancer Risk	1E-3	
CSF [$(mg/kg/day)^{-1}$]	Oral Cancer Slope Factor	see below	

COMPOUND	RfD (mg/kg/day)	DA (cm)	RAF oral	RAF dermal	CSF [(mg/kg/day)-1]	C_{sw} noncancer (mg/l)	C_{sw} cancer (mg/l)
EXPLOSIVES							
HMX	0.05	6.59E-08	1	1	NA	14,038	NA
RDX	0.003	4.79E-04	1	1	0.11	786	555.88
1,3-Dinitrobenzene	0.0001	1.06E-03	1	1	NA	24	NA
1,3,5-Trinitrobenzene	0.00005	6.33E-04	1	1	NA	13	NA
Tetryl	0.05	4.78E-04	1	1	NA	13,105	NA
Nitrobenzene	0.0005	1.88E-03	1	1	NA	110	NA
2,4,6-Trinitrotoluene	0.0005	7.12E-04	1	1	0.03	127	1,974.00
2-Amino-4,6-Dinitrotoluene	0.0005	9.89E-04	1	1	0.03	122	1,902.81
4-Amino-2,6-Dinitrotoluene	0.0005	9.89E-04	1	1	0.03	122	1,902.81
2,6-Dinitrotoluene	0.001	9.51E-04	1	1	0.68	246	84.37
2,4-Dinitrotoluene	0.002	1.15E-03	1	1	0.68	479	82.25
2-Nitrotoluene	0.01	1.91E-03	1	1	NA	2,185	NA
3-Nitrotoluene	0.01	2.00E-03	1	1	NA	2,163	NA
4-Nitrotoluene	0.01	1.95E-03	1	1	NA	2,175	NA
2,6-Diamino-4-Nitrotoluene	0.01	1.50E-03	1	1	NA	2,294	NA
2,4-Diamino-6-Nitrotoluene	0.01	1.50E-03	1	1	NA	2,294	NA
PETN		1.88E-04	1	1			
Picric Acid	0.003	4.42E-03	1	1	NA	508	NA
Ammonium Picrate	0.003	4.52E-05	1	1	NA	837	NA
METALS							
Antimony	0.0004	2.60E-03	1	1	NA	81	NA
Arsenic	0.0003	2.60E-03	1	1	1.5	61	31.47
Barium	0.07	2.60E-03	1	1	NA	14,160	NA
Copper	0.037	2.60E-03	1	1	NA	7,513	NA
Mercury	0.0003	2.60E-03	2	13.7	NA	12	NA
Zinc	0.3	1.56E-03	1.6	3.03	NA	36,537	NA

Notes:
Equations and inputs were obtained from *Risk Assessment Handbook, Massachusetts Military Reservation, Cape Cod, Massachusetts*, Air National Guard Bureau (September 1994).
RAFs were added to these equations.

Tier II Risk-Based Screening Concentrations
Ingestion and Dermal Contact with Sediment

$$C_{sed} = \frac{HI \times RfD \times BW \times AT \times 365 \text{ days/yr}}{EF \times ED \times [(10^{-6} \text{ kg/mg} \times IR \times RAF) + (10^{-6} \text{ kg/mg} \times SA \times AF \times RAF)]} \quad \text{(noncancer)}$$

$$C_{sed} = \frac{ELCR \times BW \times AT \times 365 \text{ days/yr}}{[EF \times ED \times ((10^{-6} \text{ kg/mg} \times IR \times RAF) + (10^{-6} \text{ kg/mg} \times SA \times AF \times RAF))] \times CSF} \quad \text{(cancer)}$$

C_{sed} (mg/kg)	Risk-based concentration in sediment	calculated	
HI (unitless)	Target Hazard Index	10	
RfD (mg/kg/day)	Oral Reference Dose	see below	
BW (kg)	Receptor's Body Weight	70	
AT (yrs)	Averaging Time	30	noncancer
		70	cancer
EF (days/yr)	Exposure Frequency	7	
ED (yrs)	Exposure Duration	30	
IR (mg/day)	Sediment Ingestion Rate	50	
SA (cm^2/day)	Skin Surface Area Available for Contact	19,400	
RAF (unitless)	Relative Absorption Factor	see below	
AF (mg/cm^2)	Sediment to Skin Adherence Factor	1.0	
ELCR (unitless)	Target Excess Lifetime Cancer Risk	1E-3	
CSF [$(mg/kg/day)^{-1}$]	Oral Cancer Slope Factor	see below	

			RAF		C_{sed}	
COMPOUND	**RfD (mg/kg/day)**	**CSF [(mg/kg/day)-1]**	**oral**	**dermal**	**noncancer (mg/kg)**	**cancer (mg/kg)**
EXPLOSIVES						
HMX	0.05	NA	1	0.5	187,179	NA
RDX	0.003	0.11	1	0.5	11,231	7,941
1,3-Dinitrobenzene	0.0001	NA	1	0.5	374	NA
1,3,5-Trinitrobenzene	0.00005	NA	1	0.5	187	NA
Tetryl	0.05	NA	1	0.5	187,179	NA
Nitrobenzene	0.0005	NA	1	0.5	1,872	NA
2,4,6-Trinitrotoluene	0.0005	0.03	1	0.5	1,872	29,117
2-Amino-4,6-Dinitrotoluene	0.0005	0.03	1	0.5	1,872	29,117
4-Amino-2,6-Dinitrotoluene	0.0005	0.03	1	0.5	1,872	29,117
2,6-Dinitrotoluene	0.001	0.68	1	0.5	3,744	1,285
2,4-Dinitrotoluene	0.002	0.68	1	0.5	7,487	1,285
2-Nitrotoluene	0.01	NA	1	0.5	37,436	NA
3-Nitrotoluene	0.01	NA	1	0.5	37,436	NA
4-Nitrotoluene	0.01	NA	1	0.5	37,436	NA
2,6-Diamino-4-Nitrotoluene	0.01	NA	1	0.5	37,436	NA
2,4-Diamino-6-Nitrotoluene	0.01	NA	1	0.5	37,436	NA
PETN			1	0.5		
Picric Acid	0.003	NA	1	0.5	11,231	NA
Ammonium Picrate	0.003	NA	1	0.5	11,231	NA
METALS						
Antimony	0.0004	NA	1	0	292,000	NA
Arsenic	0.0003	1.5	0.41	0	534,146	276,965
Barium	0.07	NA	1	0	51,100,000	NA
Copper	0.037	NA	1	0	27,114,286	NA
Mercury	0.0003	NA	2	0	109,500	NA
Zinc	0.3	NA	1	0	219,000,000	NA

Notes:
Equations and inputs were obtained from *Risk Assessment Handbook, Massachusetts Military Reservation, Cape Cod,* Massachusetts, Air National Guard Bureau (September 1994).

Appendix B
Derivation of Site Specific Relative Absorption Factors

INTRODUCTION

Relative Absorption Factors are necessary to adjust estimated exposure doses to be compatible with the bioavailability of doses given in the experiments that form the basis of the toxicity factors. This is required because the efficiency of COPC absorption via a particular route and matrix being evaluated varies from the absorption efficiency for the exposure route and matrix used in the experimental study that is the basis of the toxicity value. In mathematical terms, then, the RAF is calculated as:

Absorption from Environmental Media / Aborption from Experimental Administration

Based on this approach, RAFs will be media (e.g., soil, sediment, water) and exposure-route (e.g., ingestion, inhalation, dermal absorption) specific, resuling in a relatively large number of RAFs for each compound.

U.S. EPA Region I provides default RAFs for oral exposures to constituents in water or soil (1.0), as well as chemical-class specific default RAFs for dermal exposure from soil[1], but also allows for the use of chemical-specific RAFs, where data are available.

For most of the constituents of primary interest for the Training Range and Impact Area Groundwater Quality Study, data are not adequate to determine RAFs. However, information on certain inorganic compounds was found and is described below.

ARSENIC

The oral reference dose for noncarcinogenic effects of arsenic is 3E-04 mg/kg-day, and the oral cancer slope factor for carcinogenic effects is 1.5 mg/kg-day^{-1} (U.S. EPA, 1996). Both values are based on epidemiological studies that characterized health effects in a large population of Taiwanese who consumed drinking water containing arsenic. The exact form of the ingested arsenic is unknown. For the purposes of the development of the RAFs, it has been assumed that the arsenic detected is a relatively insoluble inorganic arsenic salt, such as is found in arsenic contaminated soils from mining and smelting wastes.

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1 Default RAFs for the soil-dermal media/exposure route combination are 0.5 for volatile compounds and pesticides with low sorptive capacity for soil, 0.05 for semivolatile compounds and pesticides with high sorptive capacity, and negligible (essentially 0) for inorganic compounds.

Estimation of Absorption in the Dose-Response Study

The relevant dose-response study characterized health effects in a large population of Taiwanese who consumed drinking water containing arsenic. Several studies investigating the absorption of arsenic have been performed in humans and various animal species. Human studies are sufficiently extensive to strongly suggest that close to 100% of soluble inorganic arsenic in water is absorbed from the gastrointestinal tract. These human studies are reviewed in detail here.

One direct indication of absorption of an orally administered dose of a chemical is its urinary excretion. Several studies show that urinary excretion can account for the majority of an orally administered dose of arsenic. Buchet et al. (1981a) administered aqueous sodium arsenite (NaAsO2) as a single dose to three human volunteers. An average of 45% of the dose was excreted in the urine in four days. In a second study (Buchet et al., 1981b), four individuals given 125, 250, 500, or 1000 μg As/day orally for five days excreted 54, 73, 74, and 64% of the dose in urine, respectively, over 14 days. The average urinary excretion of arsenic for the four subjects was 66% of the administered dose. Crecelius (1977) reports that approximately 50% and 80% of orally administered aqueous arsenic was excreted in urine within 61 hours by a single individual in two experiments. The results of these studies represent the minimum amount of arsenic absorbed since the balance of the dose was not accounted for.

Data for human fecal excretion of arsenic do exist. Pomroy et al. (1980) gave 6 male subjects radiolabelled arsenic acid ($[^{74}As]H_3AsO_4$) in gelatin capsules followed by a glass of water. The presence of arsenic in the body, urine, and feces was measured using a whole body radiation counter. The authors report that for the six subjects the average total excretion over 7 days was 6.1±2.8% in feces. It is not possible to determine how much of this arsenic was first absorbed and then excreted. The total recovery of arsenic (urine plus feces) was 68.4±4.0% of the single oral dose. The remaining arsenic was reported to be present in the body tissues; virtually the entire dose could be accounted for. This suggests a minimum absorption of 94% (100% - 6%) of orally ingested arsenic.

A study by Bettley and O'Shea (1975) also reports excretion of arsenic in both urine and feces. Three subjects were exposed to 8.52 mg As (as 1.25 ml of Liq. Arsenicalis B.P.) in three portions 8 hours apart on one day. They found that at most 3.5% of the dose was excreted in feces over ten days. This suggests a minimum absorption of 96%. Urinary excretion averaged 52±4% of the exposure dose over 10 days (n=3). The remaining half of the dose was unaccounted for, although small amounts of arsenic were found in blood and hair.

In the Coulson study (Coulson et al., 1935), results from two humans each ingesting two forms of arsenic are reported. Less than 5% of an oral dose was excreted in feces whether the arsenic was taken as arsenic trioxide (As_2O_3) or as natural arsenic present in shrimp. The remainder of the dose, more than 95%, was recovered in urine in three experiments where total recoveries ranged from 74 to 115%. Based on the fecal excretion data from this study, it can be estimated that at

least 95% of the ingested arsenic was absorbed. The fecal excretion data are consistent with those of Pomroy et al. (1980) and Bettley and O'Shea (1975).

Fecal excretion data from oral studies provide a minimum estimate of absorption, because it cannot be determined how much of the dose was first absorbed and then excreted into the feces. However, a study in humans injected intravenously with arsenic suggests that absorbed arsenic may be excreted, presumably from bile, into the feces. Mealy et al. (1959) administered radiolabelled arsenic by intravenous injection. Between 57% and 90% of the injected dose was recovered in urine in 10 days. Fecal excretion accounted for 1.3% of the dose after seventeen days in one individual. A second subject excreted 0.2% of the intravenous dose into the feces in one week. Both results indicate some excretion of arsenic into the feces. Virtually all of the remaining dose was recovered in the urine. Biliary excretion of arsenic has been demonstrated in rats, rabbits, and dogs (Klaassen, 1974; Gregus and Klaassen, 1986). This indicates that a portion of the arsenic found in feces in studies using <u>oral</u> dosing may have been first absorbed and then excreted.

The urinary excretion data from the oral studies discussed above provide minimum estimates of arsenic absorption ranging from 45% to 95%. The fecal excretion data suggest that, at a minimum, 95-96% of an orally administered dose of arsenic is absorbed. The study of intravenously administered arsenic suggest that biliary excretion can occur. Therefore, it can conservatively be concluded from the above studies that virtually 100% of an orally administered dose of soluble inorganic arsenic can be absorbed in humans.

RAF (Oral-Water)

The oral-water RAF for arsenic is defined as: (absorption of arsenic in humans from ingested water) / (absorption of arsenic in humans in the epidemiological study from ingested water). Since the route, matrix, and species are the same for the potential exposure in a risk assessment and the exposure in the dose-response study, the RAF is by definition 1.0. Moreover, the above results suggest that virtually all soluble inorganic arsenic administered orally in water can be absorbed from the gastrointestinal tract. Thus, it is assumed here that 100% of the arsenic was absorbed in the dose-response studies, in which humans ingested arsenic as a component in drinking water, and in the exposure route of concern - human ingestion of drinking water. Therefore, the RAF can also be defined as (100%)/(100%) = 1.0.

RAF (Oral-Soil)

The oral-soil RAF for arsenic is defined as: (absorption of arsenic in humans from ingested soil) /(absorption of arsenic in humans in the epidemiological study from ingested water). As mentioned in the introduction, there are many forms of inorganic arsenic, and these have widely varying solubilities. While it is appropriate to assume that arsenic present in water would be a soluble form of arsenic, this is not necessarily the case for arsenic present in soils. For the purposes of the development of the RAFs, it has been assumed that the arsenic present in site soil is a poorly soluble inorganic arsenic salt, such as is found in arsenic contaminated soils from

mining and smelting wastes. The oral-soil RAF for mineralogical arsenic is based on three bioavailability studies that used soil samples from mining and/or smelting sites.

Anaconda Soil Studies

The most comprehensive studies of absorption of arsenic from soil is recent work with soils from Anaconda, Montana affected by smelter activity in the area (Freeman, et al., 1993; 1995).

In the first study (Freeman, et al., 1993), rabbits were chosen as the test animal since arsenic toxicokinetics have been shown to be similar in rabbits and humans. The rat has been deemed an inappropriate human model for arsenic studies (ATSDR, 1988) because the toxicokinetics of arsenic are different between rats and humans, mainly due to the tight binding of arsenic to rat red blood cells. This binding results in elimination half-lives of arsenic on the order of days or weeks for rats as compared to half-lives of hours for rabbits and humans.
The soil used in the experiment was from the Anaconda site and was determined to be 3900 ppm arsenic. The primary mineral in the test soil was Cu_3AsS_4, an arsenic sulfide which is less soluble than arsenic oxides. The soil was screened, and only fine grains were used in the experiment. Although the use of the uniform small grain size could result in more arsenic being physically available for absorption than from non-screened soil, the grain size (100 μm) was considered to be similar to the particle size of soils found on the hands.

In this study, New Zealand white rabbits were divided into the following test groups (5/sex/group): untreated controls, single intravenous (IV) administration of soluble sodium arsenate (1.95 mg As/kg), single oral gavage (PO) administration of soluble sodium arsenate (1.95 mg As/kg), and three groups orally administered a single capsule containing increasing amounts of soil from the Anaconda site. The three soil test groups received either 0.78 mg As/kg (low), 1.95 mg As/kg (medium), or 3.9 mg As/kg (high).

Urine and feces were collected every 24 hours for five days, and samples were analyzed for total arsenic by atomic absorption spectroscopy. Clinical observations and measurements of body weight and food consumption indicated that there were no overt signs of toxicity in the animals.

The dose levels used bracket the estimated dose to a pica child (assuming a 15 kg body weight, and ingestion of 10 g soil/day), but are greater than the average child's exposure to arsenic from these soils (assuming 15 kg body weight, and ingestion of 200 mg soil/day). However, the dose level required to match the average child's exposure would have resulted in undetectable increases in arsenic concentrations in the biological samples.

For the IV dose group, an average of 77% of the dose was excreted in the urine and 8% of the dose was excreted in the feces for both sexes. The presence of arsenic in the feces indicates that absorbed arsenic can be excreted into the gastrointestinal tract, presumably in the bile. If one defines the IV dose as representing a 100% absorbed dose, it can be estimated from this data that 77% of an absorbed dose would be excreted in the urine and 8% of an absorbed dose of would be excreted in the feces.

For the PO test group (both sexes), an average of 44% of the dose was excreted into the urine. The fecal data from the oral gavage (PO) test group include arsenic not absorbed, and some arsenic that was absorbed and excreted. If one assumes that the 44% of the dose excreted in the urine represents 77% of the <u>absorbed</u> dose of arsenic administered by oral gavage, as defined by the IV data, then the total amount of arsenic absorbed from the oral gavage dose can be estimated by the following equality:

(44% dose in urine PO)/(X% dose absorbed PO) = (77% dose in urine IV)/(100% dose absorbed IV)

Therefore, solving for X, it can be estimated that 57% of the oral gavage dose of sodium arsenate was absorbed, as shown in Table 1.

A similar comparison of urinary excretion results allows for an estimation of absorption of arsenic for the soil ingestion test groups. Urinary excretion in the low test group averaged 23%. Using the same equality defined above, this would indicate that 30% of the ingested arsenic was absorbed in the low dose group. The average urinary excretion in the medium test group was 21%, indicating that 27% of the ingested arsenic was absorbed. And finally, an average of 18% of the ingested arsenic was excreted in the urine of the high test group, indicating that 23% of the ingested dose was absorbed. The average absorption of arsenic for the three soil ingestion groups is 27% with a range of 22% to 31%. Table 1 presents the data and absorbed dose estimates from this experiment.

In the second study with Anaconda soil and house dust, Freeman, et al. (1995) compared urinary and fecal excretion of arsenic in three female Cynomolgus Monkeys. Each of three monkeys were randomly cycled through the four treatments with a washout period of at least 14 days between treatments. The four treatments consisted of a single intravenous or gavage administration of sodium arsenate solution (0.62 mg As/kg body weight) or a single oral administration via capsules of soil (0.62 mg As/kg body weight) or house dust (0.26 mg As/kg body weight). Urine and feces were collected for 168 hours post treatment. They were analyzed by graphite furnace atomic absorption spectroscopy.

By the same method as described above, the amount of arsenic absorbed was estimated from the urinary arsenic of the intravenous dosing. These values are shown in Table 2.

Table 1
Data from Freeman, et. al. (1993)
New Zealand White Rabbits

TEST GROUP	ROUTE OF EXPOSURE	FORM OF As	DOSE (mg/kg)	% DOSE EXCRETED IN URINE	ESTIMATED % DOSE ABSORBED
1	Control	--	--	--	--
2	IV	Aq. Sodium Arsenate	1.95	77 (73-80)	100
3	PO	Aq. Sodium Arsenate	1.95	44 (41-47)	57 (53-61)
4	Oral Soil Capsule	Anaconda Soil	0.78	23 (23-24)	30 (29-31)
5	Oral Soil Capsule	Anaconda Soil	1.95	21 (19-24)	27 (25-31)
6	Oral Soil Capsule	Anaconda Soil	3.90	18 (17-19)	23 (22-25)

TABLE 2
DATA FROM FREEMAN, ET. AL. (1995)
CYNMOLGUS MONKEYS

TEST GROUP	ROUTE OF EXPOSURE	FORM OF As	DOSE (mg/kg)	% DOSE EXCRETED IN URINE	ESTIMATED % DOSE ABSORBED
1	IV	Aq. Sodium Arsenate	0.62	76.5 +/ 2.5%	100%
2	PO	Aq. Sodium Arsenate	0.62	69.2 +/ 3.0%	90.5%
3	Oral Soil Capsule	Anaconda Soil	0.62	15.2 +/- 4.7%	19.9%
4	Oral Dust Capsule	Anaconda Soil	0.26	25.0 +/- 3.2%	32.7%

Physiologically Based Extraction Tests

The physiologically based extraction test (PBET) is an *in vitro* test system for predicting the bioavailability of metals from a solid matrix and incorporates gastrointestinal tract parameters representative of a human (including stomach and small intestinal pH and chemistry, soil-to-solution ratio, stomach mixing, and stomach emptying rates) (Ruby et al., 1996). Arsenic bioaccessibility was evaluated in two composite residential soil samples and one composite house dust sample from the vicinity of a historical copper smelter in Anaconda, Montana.

The fraction of the arsenic solubilized in the PBET system was 50% at stomach pH 1.3 for soil #1, 32% at stomach pH 2.5 for soil #1, 44% at stomach pH 1.3 for soil #2, 31% at stomach pH 2.5 for soil #2, and 34% at stomach pH 2.5 for the dust sample. The average solubilization for these five experiments is 38%. The average for the two experiments performed at pH 1.3 was 47%. It is health-protective to use only the data from the lower pH experiments. Hence 47% is used here to represent an estimate of the human bioavailability of arsenic from mineralogic forms upon ingestion.

Other Relevant Studies

Other studies of various forms of arsenic support the conclusion that insoluble forms of arsenic are poorly absorbed. A study by Ariyoshi and Ikeda (1974) cited in U.S. EPA (1984) found 40% of an aqueous suspension of arsenic trioxide was absorbed by rabbits while rats absorbed 30%. Arsenic selenide, a highly insoluble form, was administered to humans as a fine powder and no increase in urinary arsenic was observed (Mappes, 1977). Thus, absorption in this study was probably low or negligible.

Groen et al. (1993) performed arsenic bioavailability tests in six beagle dogs with soils containing arsenic in the form of bog ore. In some parts of the Netherlands, such soils dominate the landscape. The dogs received arsenic in the form of soluble arsenic (2 mg As_2O_5) by the intravenous route and ore-containing soil (6.6-7.4 mg As) orally with food.

The experiment was performed according to a two-way crossover design, allowing each dog to be its own reference. Urine was collected for 120 hours post dosing, and it was analyzed by formation of arsine, complexation with silver diethyldithiocarbamate, and execution of molecular absorption spectrometry. Average bioavailability was 8.3 % (+/- 2.0%) compared to intravenous administration. This study cannot be used to derive an estimate of relative absorption compared to oral administration of soluble arsenic in drinking water, because the absorption of soluble arsenic in dogs is not known. However, this low bioavailability value in dogs is consistent with the above experiments.

Derivation of the RAF (Oral-Soil) for Mineralogical Arsenic

One point that is evident from the Freeman, et al. data is the difference in the oral absorption of soluble arsenic present in water in rabbits, which was 57%, and in humans, which was shown in

a previous section to be almost 100%. Therefore, it is not appropriate to apply the rabbit absorption data for soil, 27%, directly to humans. However, the data can be used if one assumes that the ratio between the absorption of arsenic from soil and from water is the same for the two species. In the Freeman, et al. (1993) study, the absorption in rabbits of arsenic from soil was 27%, and the absorption of arsenic from water was 57%. The ratio of the two values is 0.47; therefore, 47% of soluble arsenic absorbed from drinking water is absorbed from soil.

Applying this to humans, it can be estimated that 47% of soluble arsenic absorbed from drinking water is absorbed from soil in humans. Because it has been estimated that humans absorb 100% of soluble arsenic from drinking water, then 47% of the arsenic present in mineralogical form, such as in slag, would be expected to be absorbed in humans. Therefore, the oral-soil RAF for mineralogical arsenic based on the rabbit data of Freeman, et al. (1993) is:

RAF (Oral-Soil) = (47%) / (100%) = 0.47.

A similar RAF can be derived from the monkey data of Freeman, et al. (1995). The average absorption of arsenic from orally administered soil or dust was 26.3% in monkeys. The absorption of soluble arsenic administered orally in water was 90.5%. The ratio of the two values is 0.29; therefore, 29% of soluble arsenic absorbed from drinking water is absorbed from soil. Therefore, the oral-soil RAF for mineralogical arsenic based on the rabbit data of Freeman, et al. (1995) is:

RAF (Oral-Soil) = (29%) / (100%) = 0.29

The average of the RAFs based on the *in vivo* rabbit experiment, the *in vivo* monkey experiment, and the *in vitro* extraction experiment using human gastrointestinal tract conditions is 0.41. This value is used in the risk assessment as the RAF (Oral-Soil).

RAF (Dermal-Water)

The RAF (dermal-water) is used when estimating the human risks posed by dermally contacting surface water when wading or swimming or potable water when bathing. The methodology for quantitating risks posed by these exposure pathways uses a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The dose-response value for arsenic, however, is based upon an administered dose. The RAF can be used to make an adjustment of the exposure dose instead of adjusting the RfD or CSF specifically for these pathways. Thus, the RAF (dermal-water) is defined as:

(100%)/(absorption of arsenic in humans in the epidemiological study from ingested water).

Absorption in humans ingesting arsenic in water has been estimated above to be 100%. Accordingly, the RAF (dermal-water) is: (100%) / (100%) = 1.0

INORGANIC MERCURY

The dose-response value for inorganic mercury (0.0003 mg/kg/day) is a chronic RfD based on three subchronic studies in which rats were dosed with mercuric chloride either by gavage or by subcutaneous injection (U.S. EPA, 1996). This RfD is intended to be used for risk assessment of water, soils, and sediments containing unspeciated mercury. In addition, it should be used in all situations in which mercury has been speciated and found to be inorganic mercury.

The above RfD should not be used for risk assessments in which fish uptake of mercury is modeled from any source, and human consumption of mercury-contaminated fish is estimated. This is because the mercury in fish is generally in the form of methyl mercury, or some other organomercury compound. Therefore, for fish consumption, the RfD for methyl mercury should be used. In addition, the RfD for inorganic mercury should not be used whenever methyl mercury has been specifically detected and quantitated in any environmental media.

The RAFs developed in this document for inorganic mercury are applicable to soluble forms of mercury; i.e., they are applicable to most forms of inorganic mercury except mercuric sulfide. As shown below, the gastrointestinal absorption of mercuric sulfide is much lower than that of mercuric chloride, the compound upon which the RfD is based. Thus, the use of the following RAFs will cause an overestimation of the risks posed by mercuric sulfide. If it is known from site history or mercury speciation analyses that the mercury present in some environmental media is mercuric sulfide, then it may be appropriate to derive a mercuric sulfide-specific set of RAFs.

Derivation of the RfD for Inorganic Mercury

The EPA-derived RfD for mercuric chloride is a consensus value determined by a panel of mercury experts who met at EPA in October, 1987. The rat LOAEL for autoimmune effects was based on three LOAELs identified in three rat studies. In two studies mercuric chloride was given to the animals by gavage as an aqueous solution. LOAELS in these studies were 0.32 and 0.63 mg/kg-day in administered dose units. In a third study, mercuric chloride was injected subcutaneously. EPA converted the LOAEL of 0.016 mg/kg-day to units comparable to the gavage studies by assuming the following: (1) 7% of mercuric chloride is absorbed from the gastrointestinal tract and (2) 100% of mercuric chloride is absorbed when injected subcutaneously. The converted LOAEL was 0.23 mg/kg-day.

From these three LOAELS, the experts determined that 0.3 mg/kg-day was an appropriate average LOAEL from which to derive a chronic RfD. The resulting RfD is in administered dose units assuming an oral gavage dosing regimen with aqueous solutions. Thus, an estimation of absorption of inorganic mercury in rats from oral gavage is required for the development of the RAFs.

Estimation of Absorption in the Dose-Response Study

Three studies have been used to estimate the absorption of orally administered inorganic mercury in rodents. Two of these studies have defined absorption based on the residual body burden following a dose of radiolabelled ^{203}Hg. One has determined absorption based on urinary excretion data. Each of these will be discussed in turn below.

Residual Body Burden

Clarkson (1971) fed mice diets containing an unreported dose of $^{203}HgCl_2$. The body burden of mercury was measured by whole body radioactivity counting. After an initial sharp increase in radioactivity over the course of two days, whole body counts at steady state indicated that 1-2% of the daily dose was absorbed. The average of 1.5% is a minimum estimate of inorganic mercury absorption because it does not take into account the amount of the dose that was absorbed and then excreted either in urine or feces.

Walsh (1982) introduced $^{203}HgCl_2$ (46ug/kg) by gavage in aqueous solution to rats (3-6/group). At 4 hr and 43 hr post-dosing, radioactivity measurements were made on: 1) the contents of the GI tract (stomach and the regions of the intestine), and 2) the carcass after the GI contents had been removed. Radioactivity in the eliminated urine and feces were not determined. At 4 hr, 1.5% of the dose was associated with the carcass and the balance of the dose was recovered in the GI contents. At 43 hr, 2.4% of the dose was associated with the carcass and 3.3% of the dose was recovered in the GI contents. (This 3.3% may or may not be available for absorption, or may have already been absorbed and excreted.) Therefore, at 43 hr the remaining 94.3% of the dose had been eliminated from the body. The value of 2.4% then is an estimate of the body burden of inorganic mercury.

The above studies have estimated the following body burdens of inorganic mercury in rodents: 1.5% for inorganic mercury administered with food, and 2.4% for inorganic mercury administered in water. Because these values are similar, it can be assumed that the body burden of mercury administered with food is the same as the body burden resulting from exposure to mercury in drinking water. Thus, an average can be taken to estimate residual body burden of inorganic mercury after oral gavage, 2.0%.

Urinary Excretion Data

One study has reported the elimination of inorganic mercury as a fraction of the dose. This was a chronic feeding study conducted by Fitzhugh et al. (1950) in which mercuric acetate was administered in the diet to mice (20-24/sex/dose) at the following dietary concentrations: 0.5, 2.5, 10, 40, and 160 ppm. Six months into the study, urine and feces were collected for a 24 hour period and assayed for mercury content. Urinary excretion accounted for 0.5%-4.8% of the daily dose, and fecal elimination accounted for 40%-60% of the dose (values are the ranges of the averages for the dose groups).

To use the data of Fitzhugh et al. (1980) it is necessary to estimate fecal elimination from data on urinary elimination. The excretion of absorbed inorganic mercury into the urine and feces has been demonstrated by Gregus and Klassen (1986). Male Sprague-Dawley rats (4-6/group) were injected intravenously with $^{203}HgCl_2$. Urine and feces were collected, and after 4 days, fecal excretion accounted for 15.2% +/- 2.4% of the dose, and 16.3% +/- 1.4% of the dose was recovered in the urine. Thus it can be concluded from this study that fecal excretion of inorganic mercury does occur and that equal percentages of an absorbed dose are excreted into the feces and the urine.

Applying the results of the Gregus and Klassen (1986) study to the Fitzhugh et al. (1980) study, fecal excretion of absorbed inorganic mercury could also account for 0.5%-4.8% of the administered daily dose (i.e., assuming urinary and fecal excretion are equal). Therefore, it can be concluded from this study that 1%-9.6%, or an average of 5.3%, of the ingested dose was absorbed and excreted within a 24 hr time period. Alternatively, it could be concluded that the portion of the dose not excreted, i.e., 100% minus 40%-60% or approximately 50% of the dose, was absorbed. The former estimate of 5.3% and the latter estimate of 50% are minimum and maximum estimates respectively. The results of this study are difficult to interpret because the elimination data are from animals who were on a diet containing mercury for six months, and the elimination data are only for a 24 hour period. Therefore, the 24 hour determination of elimination in this study is not a complete estimation, and only minimum and maximum amounts of absorption can be identified.

Absorption Estimate

The elimination data of Fitzhugh et al. (1950) provide an estimate of the average minimum absorption of 5.3% of inorganic mercury based on excretion data. The total absorption of inorganic mercury can be represented by the amount excreted plus the amount retained in the body, which was shown above to be 2%. Therefore, the absorption estimate is 2.0% plus 5.3% or 7.3%.

This value is very similar to the value of 7% absorption assumed by EPA in the calculation of the third LOAEL used to derive the RfD for inorganic mercury. The 7% value is also supported by two additional studies described below.

Revis et al. (1992) orally gavaged five male mice with an unreported dose of $^{203}HgCl_2$ in a slurry with powdered mouse chow. After 10 days, the feces eliminated over that period and the contents of the GI tract were assayed for radioactivity. The remainder of the dose not present in the fecal sample or GI tract was assumed to have been absorbed. The value of absorption of inorganic mercury determined from this experiment, 2.1%, is a minimum estimate of absorption because the amount of the dose that was absorbed and excreted into the feces was not accounted for.

The data of Clarkson (1971) were reviewed by Clarkson (1972) where it was estimated that the measured body burden of inorganic mercury of 1-2%, when corrected for excretion, would result

in an absorption estimate of 10-15% for inorganic mercury when given orally. Quantitation of this estimate was not included in the paper, and, therefore, the results are considered here only for qualitative purposes.

The oral absorption value of 7% for inorganic mercury derived above is midway between the minimum absorption estimate of 2.1% (Revis et al., 1992) and a maximum estimate of 10-15% (Clarkson, 1972).

The value of 7.3% will be used in the RAF determinations for inorganic mercury.

RAF (Oral-Water)

The RAF (oral-water) are defined for inorganic mercury as: (absorption of inorganic mercury in humans from ingestion) / (absorption of inorganic mercury in rats from oral gavage). The estimate of absorption in rats from oral gavage, 7.3%, was derived above. Human oral absorption is discussed below.

<u>Absorption in Humans</u>

Miettinen (1973) estimated the oral absorption of inorganic mercury in humans using radioactive mercury and performing whole body counting, as well as analyzing the radiolabel in excreta and blood. Ten volunteers ingested a single, unreported dose of mercuric nitrate either in water or bound to calf liver protein (4-14 uCi). During the first four to five days, 0.17% of the dose was excreted in the urine and 85% of the dose was excreted in the feces. No difference was observed between the water and calf liver protein vehicles.

As described above, fecal excretion is a route of elimination of inorganic mercury. Thus, some of the mercury found in the feces was unabsorbed metal and some was absorbed and excreted metal. The minimum percentage of the administered dose absorbed was 15% (100%-85%) in the Miettinen study, if it is assumed that all mercury not excreted in the feces was absorbed. If it is also assumed that fecal and urinary elimination are roughly equal in humans, as they are in rats (Gregus and Klaassen, 1986), one can estimate the fecal mercury that was excreted as approximately 0.17% of the dose. This suggests that almost all of the metal present in the feces was unabsorbed. Thus, a minimum estimate of absorption is 14.8% (15%-0.17%).

The human gastrointestinal absorption value of 14.8% differs from a value of 7% that has been quoted in various articles, but which is based on the same experimental study. The Task Group on Metal Accumulation (1973) and WHO (1976) both report that 7% of soluble mercury is absorbed in humans and cite Rahola et al. (1973). This study is the original publication of work by Rahola and Miettinen. The Task Group (1973) and WHO (1976) erroneously gleaned a value of 7% from this work. The minimum absorption is, indeed, 14.8% from this experiment, as was reported in a later publication by Miettinen (1973).

Derivation of RAF (Oral-Water)

The above experimental results indicate that the gastrointestinal absorption in humans of soluble inorganic mercury (mercuric nitrate) as an aqueous solution is the same as the absorption of the same compound when administered as a protein bound species (Miettinen, 1973). This value is 14.8%.

The estimate of absorption of mercury in rats from oral gavage was derived from the literature studies presented above to be 7.3%. Accordingly:

RAF (Oral-Water): (14.8%) / (7.3%) = 2.0
RAF (Oral-Diet): (14.8%) / (7.3%) = 2.0

RAF (Oral-Soil)

The RAF (oral-soil) is defined for inorganic mercury as: (absorption of inorganic mercury in humans from ingested soil) / (absorption of inorganic mercury in rats from oral gavage).

No relevant studies cou'd be found in the literature regarding the gastrointestinal absorption of mercuric chloride, or other soluble mercury species, administered to any animal as a component of or mixed with soil. In the absence of absorption data from soil, it is assumed that mercuric chloride and other soluble inorganic mercury compounds are absorbed in humans from ingested soil to the same degree as when administered as an aqueous solution or as a protein bound species (Miettinen, 1973). This value is 14.8%. Therefore, the RAF (oral-soil) is:

RAF (Oral-Soil): (14.8%) / (7.3%) = 2.0

Mercuric Sulfide

Mercuric sulfide is very insoluble. According to Weast (1978), the solubility of alpha-mercuric sulfide is 0.000001 g/100 mL. Beta-mercuric sulfide is listed as insoluble. The only study in the literature that evaluates the oral absorption of mercury from soil used soils containing mercury primarily in the form of mercuric sulfide (Revis et al., 1990). Several experimental studies have shown that gastrointestinal absorption of mercuric sulfide is extremely low. The absorption of mercuric sulfide will be discussed here followed by a discussion of the Revis study.

Sin et al. (1983) administered mercuric sulfide to mice by gavage as an aqueous slurry. The amounts of mercury found in various tissues and organs after dosing of 300 μg Hg/week for 2-10 weeks were negligible and not statistically different from those of the controls. In the animals dosed with mercuric chloride, however, significant accumulation had occurred in kidney and spleen. The authors attribute this lack of tissue accumulation to poor absorption of the mercuric sulfide.

In another experiment, Sin et al. (1989) administered mercuric sulfide (6-324 μg Hg/g/day) to mice for four days. Very little accumulation in liver and kidney was seen compared to mice receiving mercuric chloride. These results again suggest that absorption of mercuric sulfide is very low.

Yeoh et al. (1986) fed mercuric sulfide to Swiss mice as a component of their diet (2976 μg/g) for one week. Urinary mercury was analyzed on days 2, 4, and 6, and mercury in kidney and liver was measured after one week. Mercury excretion was extremely low. On average, urinary excretion was 2.55 ng/hr. The amount excreted over seven days at this rate would be 0.4 μg, which constitutes 0.001% of the total administered dose. Mercury accumulation in liver and kidney was also extremely low. Liver mercury levels were 0.6 μg Hg/g and kidney mercury levels were 2.7 μg Hg/g. A maximum mercury burden in these tissues can be estimated assuming that 50% of the mouse body weight is liver and 50% is kidney. This maximum body burden was 41 μg, which constitutes 0.08% of the total administered dose.

Revis et al. (1990) also showed that mercuric sulfide is poorly absorbed from the gastrointestinal tract. The authors performed feeding studies with pure mercuric sulfide which showed that intestinal transit time for this compound was much longer than for mercuric chloride. With the pure sulfide, 11.3% of the dose was found in the intestinal tract and contents at 96 hours. The time estimated for the metal to clear the intestinal tract was 10 days. At this time only 0.4% of the dose had not been accounted for in the feces, thus indicating very poor absorption.

In this same study, the absorption of mercury sulfide present in soil was investigated (Revis et al., 1990). Field samples of soil from a site were mixed with a powdered mouse diet and fed to Swiss mice. Analysis of the soil determined that the mercury in the soil was present as 88% mercuric sulfide, 0.01% methyl mercury, and 7% elemental mercury. The soils were collected from different areas of the site and ranged in mercury concentration from 88 ppm to 660 ppm. The various soils were administered as 5% of the mouse diet. Seventy animals of both sexes received soil from one of seven locations for 24 hours. Animals were returned to the normal diet for 72 hours. Fecal samples were collected for 96 hours and analyzed for mercury by the cold vapor technique.

Absorption was defined in this study as the fraction of the administered dose that was not present in the feces after 96 hours. The fraction not present in feces ranged from 4-16% for the different experimental groups, with an average being 9.1 +/- 4.3 (n=70). As shown above, however, fecal excretion is a route of elimination for inorganic mercury. Thus, it is not possible to determine what fraction of the amount present in the feces was unabsorbed metal and what fraction was absorbed and excreted metal. In addition, as shown below, mercuric sulfide has a long transit time in the intestinal tract. Thus, some material present in the animal's body is unabsorbed metal that has not made its way through the intestinal tract. The authors suggested that the mercury containing soil would also have shown a lower fraction <u>not</u> present in the feces if the animals had been followed for 10 days instead of 96 hours. Thus, the absorption estimate from this experiment is certainly an over-estimate.

All of the data presented above strongly suggest that the oral absorption of mercuric sulfide and mercury from mercuric sulfide containing soil is extremely low. Thus, the RAF (oral-soil) for soils containing primarily mercuric sulfide would be close to zero. However, the chemical species of mercury are not usually known for environmental samples. It is not known to what extent the presence of soil may retard the gastrointestinal absorption of soluble inorganic mercury compounds. Accordingly, the RAF (Oral-Soil) derived above must be applicable to mercury species other than mercuric sulfide. In the absence of data for soluble mercury species, it has been assumed that gastrointestinal absorption is the same for a soil matrix and for aqueous oral gavage. In this case the RAF (Oral-Soil) is 1.0. If it is known that mercuric sulfide or another insoluble compound is present, a site-specific RAF can be derived based on animal feeding studies with site soil or extractability studies with acidic solutions that mimic the conditions of the stomach.

RAF (Dermal-Water)

The RAF (dermal-water) is used when estimating the human risks posed by dermally contacting surface water when wading or swimming or potable water when bathing. The methodology for quantitating risks posed by these exposure pathways uses a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. The RAF is used to make an adjustment of the exposure dose.

Thus, the RAF (dermal-water) is defined as: (100%) / (absorption of inorganic mercury in rats from aqueous gavage). This RAF is derived using the estimate of rat absorption of 7.3% from Clarkson et al. (1972). Accordingly, the RAF (dermal-water) is:

RAF (Dermal-Water) = (100%) / (7.3%) = 13.7

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ZINC

The oral dose-response value for zinc (3.0E-01 mg/kg-day) is based on a human study with zinc sulfate in the diet. Thus, the RAF (oral-diet) is 1.0. The mean absorption of zinc from diet in ten humans has been determined by Sandstrom et al. (1987) as 33% (22-46%). The absorption of zinc from drinking water was determined in humans by several workers: 56% (Dinsmore et al., 1985), 42% (Milman et al., 1983), 58% (Farah et al., 1984) and 55% (Valberg et al. 1985). The mean of these four values is 53%. Thus, the RAF (oral-water) is 53%/33% = 1.6. The gastrointestinal absorption of zinc from soil is assumed to be identical to that from diet. Thus, the RAF (oral-soil) is 1.0. A recommended default value of 0.1% (EPA, 1992) was assumed for dermal absorption of zinc. Thus, the RAF (dermal-soil) is 0.1%/33% = 0.003.

The RAF (dermal-water) is used when estimating the human risks posed by dermally contacting surface water when wading or swimming. The methodology for quantitating risks posed by this exposure pathway uses a chemical-specific permeability constant that estimates the rate at which the chemical passes into and through the skin from an aqueous solution. By definition, the dose estimated by this procedure is an absorbed dose. Most dose-response criteria, however, are based on administered doses. An adjustment is necessary to account for the absorption in the dose-response study. In order to use consistent dose-response criteria across all exposure pathways, the RAF is used to make an adjustment to the absorbed dermal dose, instead of adjusting the dose-response criteria. Here, the RAF is defined as (100%)/(estimated absorption in the dose-response study). For zinc, the RAF (dermal-water) is 100%/33% = 3.03.

Inhalation 1.0

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